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SOLID FOOD FOR BABIES

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'As we learn more (about infant nutrition) our present ideas will undoubtedly change, and the infant feeding of 1950 *will probably be little like that of* 1920' (Lewis Webb Hill, 1922).

There is a 'new look' in infant nutrition; one that would have made Czerny and Finkelstein gasp, Rotch and Truby King shudder, with its disquieting implication that babies are hardy creatures, not fragile, with digestive organs that are far from inadequate, indeed phenomenally good, and with its tacit admission that the artificial feeding of infants is a wonderfully easy matter, needing virtually no skill at all, only care.

Until recently, changes of fashion in infant feeding were so insidious as to be practically undetectable. It took some 3,000 years before it was accepted that solid supplements could be included in the baby's diet, not at the age of 3 years, but at 3 months. In this century, however, things have been gathering momentum, and in the last 10 years it has been accepted that solid food can be given to babies soon after birth. Thus, fable has become fact: It was some 2,000 years ago, according to legend, that ben Sira, newly-born, disdained his mother's breast, and demanded solid food instead.[1]

The addition of solid foods to the diet of infants and children has a venerable and vexatious history which is only now drawing its many chapters to a close. The background, of course, is one of milk feeding, either by maternal breast, wet nursing, or dry nursing ('hand' or 'artificial' feeding). Against this background must be sketched first the addition of various gruels and porridges—from ancient usage the first to be added to a hitherto all-milk diet. Intimately bound up with the use of gruels is the subject of the eruption of the teeth, and the relationship of teething to weaning. Finally there is to be considered food other than gruels—these being virtually newcomers to the field of infant nutrition.

From the earliest recorded times until the Renaissance, infants were breast-fed or wet-nursed until perhaps 2 or 3 years of age. Such feeding often virtually excluded any other form of nourishment. In biblical times nursing lasted up to 3 years (2 Macab. 7 : 27). The Koran requires 2 years of breast feeding, though it does sanction earlier weaning (Surah 2, verse 233). Shakespeare's Juliet was nursed for almost 3 years (Act I, sc. iii).

CEREALS

As a general rule, some time during the 2nd half of the first year, thin gruels were cautiously added.

Hippocrates (460 B.C.)[2] advised that for the first 6 months only breast milk was to be used. The same opinion was voiced by Soranus (2nd century A.D.)[2] who added that after 6 months bread crumbs soaked in water, milk or wine might be added. The practice of soaking bread in meat and vegetable broth has been ascribed to the earliest Greeks,[3] and indeed, the advice on the use of bone and vegetable broth still found in modern publications, is no more modern than Galen.[4] In ancient Egypt supplementary foods were begun after 6 months,[5] and the Arabic physician Avicenna (11th century)[3] recommended the use of bread soaked in water or sweetened milk. Jeremiah laments: 'The children and the sucklings swoon in the streets of the city. They say to their mothers, Where is corn and wine?' (Lament 2 : 11-12).

Such gruels as indicated above continued to be in general use in Europe until the Renaissance, when, with the massive increase in bad wet-nursing and in disastrous 'dry'-nursing (animals' milk), attempts were made to nourish infants on thin gruels long before the age of 6 months, and indeed such gruels played a most prominent part in infant nutrition even up to about 1850.[2,3] In Europe such gruels were known as 'pap' and 'panada' and were usually given to infants with pewter feeding vessels of quaint appearance. Pap and panada consisted of bread crumbs, barley, rice, oatmeal, flour, or biscuits soaked or cooked in various fluids such as milk, water, broth, beer or wine. When of a formed consistency, pap was often pre-chewed by the mother or nurse before being placed in the infant's mouth—a procedure recommended by such authorities as Peachey (1697),[3] and before him, Avicenna.[2]

Much was written about pap and panada after the Dark Ages when such cereals were offered at any time from one year of age to one month, or even earlier. Ambrose Pare (16th century)[3] prescribed such 'solids' at 10 days. In liquid form large quantities of pap were fed to small infants, almost to the total exclusion of breast or animals' milk—and the results were catastrophic.

Metlinger (15th century) advised thin gruels when the nurse was ill or had little milk. Like Avicenna, he gave mixtures of bread, water, milk and sugar. He wrote: 'Children are to be nourished on gruel and milk until they cut their front teeth'.[3,6] On the Continent, in the 18th century, the use of pap from birth[2] (especially milk with barley flour) was widespread. By 1800 there was in use, from birth, not only pap and panada, but also barley water, soup, beer, rice, oatmeal and tapioca.[3] One of the rationalizations for the early use of these foods was that their introduction before 6 months helped to establish the habit of giving solid foods (Alexander Hamilton, 1792).[4]

A little consideration will make it clear that the use of cereals after 6 months, as well as virtually from birth, is very much in evidence today. There are many babies who are given no porridge until they are 6 months old, while other infants have cereals placed in their bottles during the first few weeks—even days—of life.

Among some of the Bantu people feeding practices show even greater similarity to those current in Europe 200-300 years ago. On the one hand, infants may be fully breast-fed and not get cereal supplements for 6 months or longer, and on the other hand, some of them may be brought up, become malnourished and die on a diet of pap—porridge cooked in water or black tea, with an occasional addition of

overdiluted milk, or perhaps a desultory suck on an inadequate breast.

With the emergence of safe milk in the late 19th century, coupled with the production of reasonably satisfactory glass feeding bottles, the habit of very early gruel feeding receded beyond the 6-month barrier. Any solid supplement was considered unsuitable much before a year, though in deference to tradition, cereal continued as the first addition to an all-milk diet.

From about 1850, when safe milk was introduced, until the turn of the century, big business in baby foods made its appearance and flourished. Understandably, the products available for sale consisted of various types of milks, of cereals, or of mixtures of the two. By the early 1900's there was a bewildering array of proprietary infant foods. Some of the better known brands were: Allenbury's, Liebig's, Borden's, Prince of Wales', Mellin's, Horlick's, Savoury and Moore's, Robb's, and Robinson's food, and the better known milks included: Swiss milk, Biedert's cream, Gartner's milk, von Durgern's renneted milk, Szekely's casein-free milk, Voltmer-Lahrmann's pancreatized milk, Backhouse's milk, Mortis' whey milk, Vigier's humanized milk, Lehndorf and Zak's dialized milk, Feer's milk and Schloss' milk.

In 1884 Eustace Smith[7] permitted the use of 'farinaceous' foods at 6 months. In the 1920's starchy foods were generally authoritatively recommended to be begun at 9 or 8 months,[8] in the 1940's at 6 months,[9] and in the 1950's at 3 months.[10]

WEANING AND TEETHING

The introduction of solid food to an infant's diet cannot be understood without reference to the times of dental eruption. Weaning is intimately bound up with teething, and the subject of the appearance of the deciduous teeth dovetails nicely into the saga of supplementary foods.

In earlier times the matter of weaning was of considerable importance, especially the termination of weaning. Today it is the beginning of weaning that is of interest and its termination is unimportant. Indeed, the very meaning of the term has altered. In former days weaning indicated the time for the introduction of food other than breast milk. Today weaning means the addition of 'solid' food to a hitherto all-milk diet—cow's as well as breast milk. Indeed 'weaning' has become a redundant term; we can quite easily dispense with it entirely. Weaning means no more than the introduction of 'solid' food to a baby's diet.

But there were times when weaning had a pronounced and resounding end. Weaning was a long discipline, beginning usually when the first deciduous teeth appeared, and ending with the eruption of the last primary teeth. Until such time the principal item of diet was breast milk. Indeed that is precisely why the deciduous set are termed 'the milk teeth'. It was only after the final milk teeth had appeared—somewhere between 2 and 3 years—that a child was considered sufficiently mature to deal with an 'adult' diet.

The last feed was often a public occasion ('And Abraham made a great feast the same day that Isaac was weaned' Gen. 21 : 8). The nipple was smeared with pepper, aloes or some other vile substance and the breast offered to an unsuspecting child. Puzzled and enraged, the child recoiled from the breast. Once more, perhaps, the breast was offered, and weaning was complete! Such dramatic weanings were common in Europe until as late as 1800.

It seemed self-evident that solid food should only be introduced when there were teeth present to deal with it, with the result that from earliest recorded antiquity we have advice about introducing cereals at about 6 or 7 months.

Soranus wrote that supplementary foods (bread crumbs soaked in various fluids) could be given when the first teeth appeared, and added that it was unnecessary to delay weaning in girls until they were a year old![3] Hippocrates suggested that food other than milk should be introduced so as to coincide with the appearance of the teeth.[2]

Such views on the most suitable time for introducing solids have been echoed down the centuries until about 1950. Thereafter a new criterion has been suggested for the introduction of solids: the ability of the infant's tongue to transfer food from the front of the tongue to the back of the pharynx. This criterion[11] has permitted an aura of respectability to the giving of solid food at 3 months.

But from the 15th to the 18th century, when there was a rapid spread of disastrous wet-nursing and hand-feeding, the only alternative was to offer greater quantities of liquid pap and cereals at earlier ages—even soon after birth—without reference to the appearance of the teeth. While numbers of writers held that pap should only be introduced when the front teeth appeared (Underwood,[12] 1784), probably the majority of infants in Europe from the 15th to the 18th century were fed (and usually died) on a highly dilute pap (with occasionally sufficient breast or animals' milk) from earliest infancy—as advocated by Metlinger (1473) and others.[6]

Now, although by 1800 there was general agreement that solids should not be given until 6 or 7 months (some more conservative writers held that it was wise to wait until 4 incisors were through), there was a distinct difficulty about using dental eruption as a yardstick for weaning—a difficulty well known to all scholars. For one thing, teeth might not appear for a year or longer. For another, there was widespread knowledge concerning the Roman hero Marcus Curius (250 B.C.) who, having been born with teeth, became known as Marcus Curius Dentatus.[13] When should one begin cereal feeding under circumstances like these? And he was not the only child born with teeth—among the more famous examples were Richard III of England and Louis XIV of France.[13]

By 1800 it was also realized that milk was not a perfect food and that solids too were important for infant nutrition. Armstrong (1771)[2] had in fact stated that it was unnecessary and undesirable for breast feeding to last until all the teeth appeared, and that 18 months' nursing was sufficient. Underwood (1789)[3] was content with 12 months. By 1850 it was clear that the end of weaning had nothing whatever to do with the teeth, and that a year-and-a-half's nursing, or even a year's nursing was sufficient.

If, however, the end of weaning was not associated with teething in 1850, the beginning of weaning still was for at least another 75 years. In 1922 Eric Pritchard wrote that at 9 months 'a certain amount of solid food is indicated owing to the eruption of the teeth'.[8]

By the 1950's a certain amount of solid food was permitted at 3 months by virtue of the new criterion that the tongue was able to transfer food to the pharynx.[11]

Perhaps by the 1960's solid foods will be permitted even earlier, based on the criterion that infants, like adults, are after all human?

NON-CEREAL SOLIDS

It is probable that foods other than gruel played some small part in the nutrition of young children in earlier times.

Among the dynastic Egyptians, vegetables were introduced after some 6 months of breast feeding.[5] Fruit and vegetables, and perhaps even meat and fish were probably used sparingly until the completion of weaning. The *Bible* laid down detailed laws on what might be eaten among fish, flesh and poultry, but oddly enough, eggs as an article of diet is not mentioned in the Pentateuch.

Oribasius, a 4th century Turkish physician, best known for the excellence of his unacknowledged plagiarism of Soranus, advised against the use of meat for small children. 'Meat is bad' he wrote, 'and also thick soups'.[6] Subsequent opinions on meat for babies remained practically unchanged for the next 1,500 years. Metlinger, in the 15th century, wrote that meat caused worms to grow, especially if it was 'strong' meat, and given in too great a quantity.[6] He permitted a small amount of finely-cut well-cooked lean meat during the second year.[3] In the 16th century it was common practice to give minced poultry at 15 months and red meat at 2 years (Guillemeau).[3]

Peachey (±1700) gave chickenbroth after the completion of

weaning, followed by 'flesh of easy digestion'—if necessary, pre-chewed by the nurse or mother.[3]

In the late 18th century, a 'Ladies' Journal' published in America wrote an account of what must have been the general views on infant feeding at that time. The best foods for children, according to this article, were milk, bread and cereals, while a small beer (!) was also desirable for boys. Plain dry bread was the ideal food. Fruit was unsuitable for small children, and meat might be tried at 3 or 4 years, and even thereafter not more than once a day.[2]

'Since they overstimulate the appetite', stated the Journal, 'spices or salt are not to be used. Fruit is not good. Children, though, are not to be forbidden all fruits, for such a course would result in bribery of servants or some other surreptitious device for obtaining them'.[6]

In England, in 1742, Walter Harris complained that some children sickened and died as a result of getting meat too early;[2] but it is probably unlikely that mothers were as terrified of feeding children as were the authoritative writers. Caufield notes the remarks of the American colonial mother who was proud and happy to feed her 8-months-old son his usual dinner of fat pork and Indian cake.[14]

About 1880 Eustace Smith wrote that he gave no meat until well after a year,[7] and until about 1925 babies were considered to have such delicate digestions that nothing but milk and cereals were to be given before the age of a year. Thereafter egg yolk might be tried, meat offered at about 2 years, and bananas even later.

But the picture is not quite so bleak. Every now and then an enlightened soul dared to give some solid food at an early age. Paul of Aegina (7th century)[6] offered eggs shortly after 6 months. Vallambert (1565) added egg yolk to pap for use at 3 months.[4] About 1770 the redoubtable Armstrong tried broth and beef tea at 5-6 months and minced chicken a few months later,[2] and the remarkable Dr. Cadogan fed babies of 6-8 months on bread and butter, vegetables and roots of all description, raw, stewed or baked fruit, and even flesh-meat.[2,3] In 1773 Fordyce[3] complained that some infants were being fed on meat, strong broths, buttered rolls and muffins even before their teeth had appeared. By 1800 it was not uncommon for egg yolk to be used in the making of pap.

In 1825 potatoes might have been offered occasionally at 9 months of age.[15] Towards the end of the century Eustace Smith gave the yolk of an egg at 10 months as well as weak broth of beef, veal and mutton.[7] A light pudding was allowed at a year, and meat only later.

The first quarter of the 20th century was one of great fear of feeding a baby at all. Overfeeding was the great bogey, and virtually all foods were accused of producing 'intolerance'. In 1922, therefore, it was reasonable to expect an authority on infant feeding of the calibre of Hill to complain about the perilous extremism of some of his contemporaries who were offering potatoes and other vegetables as early as at 8 months. He writes: 'In this country, in recent years, the practice of giving vegetables to small babies has been pushed to an extreme, and we often see babies of 7 or 8 months who are taking large amounts of potato and other vegetables'.[16]

Nothing daunted, in 1924 Jundell of Sweden proceeded to feed 6-month-old infants on minced meat, fish, eggs and vegetables.[17] Such feeding at an early stage was given a great impetus by the discovery of the importance of vitamins, and in the 1930s solids were occasionally allowed as early as at 4 months. Glazier[18] (1933) wrote an account of the introduction of solids at 2-3 months. Ten years later, while most respectable paediatricians still advised solids no earlier than 6 months, there was an astonishing report by Stewart who fed gruel by spoon from the 3rd day. That was nothing remarkable of course, but he also fed fish, sardines, carrots and peas in their skins to infants of 1-2 months.[19]

MODERN RADICALS

During recent years, while conservatives such as Sheldon (1955)[20] are content to wait until 6 months, and liberals usually begin at about 3 months, the radicals have given solids just about from birth—even to premature babies.[21] The radicals are enthusiastic about it, believing that very young babies fed on meat are healthier, happier, sleep better and have fewer colds and higher haemoglobin values.[22]

Since 1946 Gough in Quebec has been giving orange juice to 7-day-old infants, followed by cereal and then at 12-14 days by fruit (including pears and prunes). At 3 weeks the baby is given meat and whole cow's milk. Thereafter vegetables, egg yolk, more cereals, egg white and more meat are given.[23]

Not to be outdone, Sackett of Miami (1956) gives cereals on the 2nd day, vegetables at 10 days, meat at 14 days, fruit at 17 days—at which time he drops the midnight feed and tries to institute a regime of 3 meals a day. The babies are then given their proper meals (rather than drinks) at 6-hourly intervals. He writes: 'An attempt is made to convince the mothers of the wisdom of making the babies conform to adult lives, rather than making the family adjust to the demands of the baby.'[24] This is certainly behaviour therapy with a vengeance!

In the United States most babies cared for by the younger paediatricians now start on solids before 3 months. The much advertised cereals are often given first—either by spoon or in the bottle. The principal reason for beginning with cereal is that it is traditional. Modern cereals are derived from the pap of earlier days. Tradition often has a great deal to recommend it, but it seems that in this instance tradition is not based on a firm foundation. While newborn babies are not generally lacking in digestive capacity, the one enzyme that is not infrequently deficient is amylase—for the digestion of carbohydrate.[25]

Since 1955 I have been feeding solids to very young babies. I have begun as early as 10 days on the arrival of the infant from the nursing home, or as late as 3 months. Not all babies take to it and a great deal depends on how the other children in the family were fed and on the opinions of parents, grandparents, relatives and friends.

A few infants are unhappy on solids begun before about 3 months. Others love it and will take virtually anything placed in their mouths. I have seen 3-week-old infants gorge themselves with half a banana and sigh with contentment. The large majority of very young infants take readily to solids, but many of them have pronounced dislikes—often for eggs and for the baby meats produced (in glass containers) by various manufacturers. Sometimes heavy sweetening of these foods will make them acceptable, or even using a different brand of 'chicken and vegetable' may prove successful.

Somewhere about 6 weeks many mothers become restless and bored with the same daily routine, and long to add something else to baby's diet. Frequently they pluck up sufficient courage to try a bit of chocolate, and thereafter further additions are easy. Indeed, somewhere around 7 or 8 weeks, when many infants sleep through the night, it is not at all difficult to adjust the diet to coincide with 3 meals a day—especially if the infant has been taking solids well for a few weeks earlier.

I normally begin feeding solids with bananas, or occasionally with chocolates. These are given towards the end of a breast or bottle feed. The solid is placed on the back of the tongue by means of a small teaspoon. Bananas have a number of advantages as an initial food: (1) They are seldom refused (the great majority of infants love bananas) (2) they are very easy to mash and to give by spoon and (3) mothers gain confidence in noting how readily their

infants take to bananas and have no qualms about offering other solids.

Most babies prefer their solids towards the end of a feed, and the solids can be given 3 times a day. There is no need to proceed with fear and trepidation. Indeed, there is no reason whatever for not giving a whole egg for breakfast if the infant obviously likes it. Within a week of giving solids for the first time, 10 or more different articles of diet may be introduced.

Apart from bananas and chocolates, other foods which may be given (singly or mixed) include custard, ice cream, soups and juices, apple sauce and various canned fruits such as peaches (especially liked), pears, apricots, guavas and prunes; also vegetables like squash, peas, pumpkin, carrots, pawpaw and avocado pear. All these are more cheaply prepared in the home rather than purchased ready-made. Also suitable for babies, but particularly so at 4-6 weeks, are whole egg, strained liver and meats (best purchased rather than prepared—the texture of prepared meats may not be sufficiently fine), boiled fish, sardines (easily mashed with milk), peanut butter (with added milk) and finely grated cheese (in milk). Finely pounded bacon can be made semi-liquid with the addition of egg yolk so that there is no need to deny babies the traditional breakfast of bacon and eggs! Less important foods are potatoes, rice, jelly, jam, puddings, semolina, the much advertised cereals and finger biscuits mashed in milk. 'Marie' biscuits are unsuitable as bits tend to stick to the palate.

Solid foods are poorly named; most of them, in the form given to babies, are more liquid than solid. Milk itself is a unique solid—this is quite obvious when vomiting has occurred some time after a feed. Gastric enzymes turn milk into solid clots before it is digested. Milk is a solid disguised as a liquid, so that in any event babies are getting solids right from birth.

Solids must share 3 properties with milk: (1) They must be sterile, or at least not subject to ready contamination, (2) like the curd of milk, the texture of the solid should be as fine as possible and (3) the vehicles used for solid feeding must be sterile. These include cups, saucers, knives, forks and spoons and they are much easier to keep sterile than bottles and teats.

ADVANTAGES AND DISADVANTAGES OF FEEDING SOLIDS

Advantages

It seems to me that the principal advantage of the use of solids is the demonstration to the mother that her baby is not a frail, fragile creature, that it does not have to be handled with needless restrictions, and that fantastic and intricate formulas derived from various emasculated milks are not necessary in baby feeding. The use of solids illustrates the truism that babies are human. The advantages of giving solids before 3 months are exactly the same as giving them before 6 months . . . 12 months . . . 3 years. . . .

My impression (and it is *only* an impression) is that infants who are used to a wide variety of foods very early in life give little trouble by refusing food later in the first year.

Another impression is that babies fed on solids are happier, sleep better (because the emptying time of the stomach is longer?) and that they are less subject to colic than those fed only on milk.

Another advantage is that it is a good thing to teach an infant the use of the spoon. Moreover, babies getting a wide variety of solid foods do not need vitamin supplements. These substances are not always innocuous. There is no substitute for good food, even in earliest infancy.

Disadvantages

Strenuous objections to early feeding of solids have been voiced. The principal objection is that of precipitating allergic reactions. Thus far there is no evidence to support such a fear,[26] and I doubt that it has any validity. However, this is a subject about which we need more facts and less opinions.

The danger of choking when an infant is given solids comes to mind, but I have never found this a factor to be seriously considered. I also do not believe that giving solids as such interferes with the incidence and duration of breast feeding.

The question of overloading of the kidneys, must, however, be seriously considered. If too many solids are given, there may be insufficient water intake to deal adequately with the solute load,[26] especially in bottle-fed babies. If we assume that the infant's kidneys can only deal with a total solute load of about 700 m0s/litre[27] (adult maximum about 1,400 m0s/litre), the water available for the excretion of a large solute may be insufficient, especially during hot weather or when diarrhoea is present, and a state of hyperosmolarity may result. In this respect it is important to note that babies do not differ from adults. They require water, especially if they are feverish, or if they are losing water by vomiting, diarrhoea, perspiration or respiration. The important factor in the feeding of solids to very young babies is certainly not quantity, but variety.

Finally there is the *canard* of food tolerance, especially fat intolerance. There is a widespread fear among lay people of giving any kind of solid food to babies, and this fear is nothing more than a result of earlier views on the delicate nature of babies and on the intricate skill which was supposedly necessary for their correct feeding—skill incidentally, which was little more than an exercise in starvation. Ingrained views on intolerances, dyspepsias and indigestions are responsible for the fear of feeding infants not only with solids, but also with undiluted cow's milk. Radical views are heresy to those whose first query is 'but isn't it too strong'? The whole subject of digestion and indigestion in infancy has been dealt with most admirably by Gans,[28] in a forthright attack on outmoded concepts of feeding. His paper should be compulsory reading for any student who aspires to feed babies intelligently. Suffice it to say that very young infants have most efficient digestive organs, and that indigestion (except during intercurrent illness) simply does not exist. If intolerance to food does occur, the intolerance is likely to be in the kidney, not the gut. Fat absorption and utilization in infants are especially good.

The Committee on Nutrition of the American Academy of Pediatrics has investigated reports on the early feeding of solids and, on the evidence thus far available, has not yet been convinced of the superiority of such a practice, and has taken a stand on the matter which might be termed one of negative neutrality. The Committee can detect ' ... no nutritional superiority or psychologic benefit from introduction of solid foods ... prior to 2½ to 3 months of age'.[26]

THE FUTURE

Having scrutinized the past and examined the present, let us look into the future.

There seems little doubt that the giving of food to infants is no different to the giving of food to adults. Age is no criterion. Very early feeding of solids will become increasingly widespread, but the novelty will wear off, just as the novelty wore off after beginning weaning at a year, 9 months ... 6 months ... 4 months ... 3 months. The opinion on early feeding will probably be that it is generally beneficial, and in 10-15 years we might note some questions and answers as follows:

Mother: Shall I give my baby solids?

Doctor: Yes, if you want to.

Mother: When can I begin?

Doctor: Whenever you feel like it; it's not an important question.

Mother: What foods can I give?

Doctor: Anything you wish, as long as the food is clean and finely mashed.

Mother: At what times should I give the solids?

Doctor: Whenever you feel like, or when you think your baby would like it.

Mother: How much at each feed?

Doctor: As much as the baby wants, but variety is more important than quantity.

Such replies might be exasperating in 1959, but will be perfectly acceptable in 1969—indeed the answers will not even be given, nor the questions asked, for these matters will be common knowledge and everyday practice.

SUMMARY

An account is given of the feeding of solids to babies, beginning with the views of earlier days, present practices and tendencies, and a glimpse into the future.

Gruels of one type or another have always been used as the first solid of weaning, and their introduction is intimately bound up with the times of the appearance of the deciduous teeth. Solids other than gruels have been used sporadically since antiquity and are at present ousting the cereals as the first food to be given during weaning.

Solids are often given during the first few weeks of life, and in general this practice is beneficial.

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QUESTIONS ANSWERED : ANTWOORDE OP VRAE

BACK ACHE WITHOUT NEUROLOGICAL SIGNS

Q—*One of the trying problems in the life of the general practitioner is the problem of severe back ache without neurological signs in healthy young men between the ages of* 20 *and* 40. *How should a country practitioner approach and treat this condition?*

A—Acute back ache in healthy young men is certainly a common condition. Where there is a history of severe strain or trauma the aetiology of the condition need not be in question, but often, there is no such history. Infections, notably tuberculous caries, and occasionally ankylosing spondylitis or, even more rarely, secondary neoplasms or myelomatosis, may cause back ache; however, these conditions represent only a very small percentage of cases with acute back ache. In view of this, it may be well to consider whether there is any common factor that would make young healthy men liable to some type of weakness or strain of the back.

The answer to this question may lie in our way of life, which so frequently includes the driving of cars and lorries, with the back flexed for long periods, or sitting at desks with the back in anything but the normal upright position—interspersed with occasional rapid bending to pick up heavy cases and other objects (including instrument cases in our own profession). It is generally agreed that the aetiology of the great majority of cases of acute back ache come into the mechanical group, and that, whether a small bulging disc or a disorder of the apophyseal joints is the causal agent, the treatment is essentially the same.

In the very acute case the patient must be put to bed, preferably with boards under the mattress. It is important that patients should be told how to lie in bed—it is useless for them to go to bed with back ache if they lie curled up or if they sit propped up with the back flexed, to read the paper and greet their friends. They can be allowed up for toilet and it is better for them to have their meals sitting in a high hard chair, with a cushion behind the back, than in bed. After about 10 days it should be apparent whether this treatment has helped. If it has not, then further investigation is needed and the possibility of wearing a support for the back should be kept in mind.

In less severe cases all that may be needed is a firm bed at night (but again with the proviso of sleeping in an extended position), and avoidance of the flexed position while driving a car or sitting during the day, by the use of a pillow in the small of the back. After the acute stage is over, not only must the patient avoid the lifting of weights, but he should take serious steps to strengthen his back muscles by leg-raising exercises while his back is kept in the neutral position. There is no doubt that the great majority of cases of acute back ache can be dealt with adequately if the doctor and patient consider what positions in the 24 hours of the day might produce strain or pressure on sensitive structures of the back, keeping in mind that a mild strain over 3 or 4 hours may be as damaging as a more severe acute strain.

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE ROOKGEWOONTE: BENADERING TOT 'N BELEID

Die rookgewoonte het waarskynlik ontstaan onder die Rooihuide van Amerika, wat die tabakplant gebrand het as 'n offer aan die groot gees, en die rook ingeasem het—die eerste rokers dus. Columbus het drie dae na die ontdekking van die Amerikas op 15 Oktober 1492 aantekeninge gemaak van die gewoonte.[1] Sedertdien het baie skrywers die pen opgeneem vir en teen die gewoonte. In Suid-Afrika het die Hollanders die gewoonte onder die Hottentotte gevestig, wat dit weer aan die Boesmans geleer het. Teen 1705 het alle rasse en bewoners van Suid-Afrika die gewoonte geken. Die geskiedenis van die rookgewoonte en sy verspreiding in ons land, asook die evolusie van die pyp in Suid-Afrika[2] bied aangename leesstof oor 'n koppie koffie en 'n pyp, saans voor die vuur.

Of ons as geneeshere rokers is of nie, sal van ons persoonlike omstandighede en beskouing afhang. Ons moet egter besef dat ons ook 'n plig teenoor die publiek het en 'n plig teenoor die sieke in besonder. Te veel ekonomiese faktore speel vandag 'n rol in die tabaknywerheid, sodat daar nie gehoop kan word op wetgewing om die gewoonte te bekamp nie. Waar ons egter 'n soutvry dieet in sekere omstandighede kan voorskryf sonder om dit self te volg, betaam dit ons om sekere aanduidings vir verbod op hierdie gewoonte te bepeins. Ook moet ons 'n kode van gedrag bepaal ten opsigte van die publiek vir wie ons as raadgewers optree, en vir pasiënte by wie die sukses van ons behandeling afhang van hulle uitvoering van ons opdragte.

Die gesonde persoon wat om advies vra. Ons kan vandag met beslistheid sê dat daar 'n verband bestaan tussen die rook van sigarette en longkarsinoom, of, in die woorde van 'n Engelse skrywer: ‚Present evidence of an association between cigarette smoking and lung cancer is so strong as to be considered proof within the everyday meaning of the word'.[3] Die rol van interne faktore word nie ontken nie, want alle rokers kry nie longkanker nie en longkanker kom wel soms voor onder nie-rokers, maar die kanse om hierdie siekte te kry (wat vandag een van die belangrikste oorsake van kankersterftes is), word baie verhoog deur rook. Pyp- en sigaarrokers toon ook 'n veel hoër voorkoms van die siekte as nie-rokers, hoewel nie soveel meer as die sigaretroker nie.[4]

Die rookgewoonte word skynbaar nie toevallig aangeleer nie, maar wel as gevolg van sekere persoonlikheidseienskappe en fisiologiese kenmerke eie aan die roker.[5] Wat die publiek betref, moet ons dus pleit vir die aankweek van matigheid, net soos in die geval van drankgebruik, want die hoeveelheid tabak wat verbruik word hang saam met die waarskynlikheid van voorkoms van longkanker. Hoe minder ons per dag rook, hoe kleiner word die kans om longkanker te kry.[4]

Die rookgewoonte hou ook groot potensiële gevare in vir die hart en bloedvaatstelsel. Onder jonger mense met miokardiale infarksies is die mortaliteit hoër by rokers as by nie-rokers.[6] Verder is dit ons morele verpligting om ons aandag op die jeug toe te spits en die gewoonte onder hulle aandag te bring as 'n skadelike gewoonte. Die bedreiging van chroniese bronchitis en emfiseem, die waarskynlikheid van longkanker, die verband met vaskulêre siektes soos trombo-angiitis obliterans, word daar gelaat. Die elektro- en ballisto-kardiografiese bevindings voor en na die rook van sigarette toon dat daar wel deeglik kardiovaskulêre veranderings is in normale persone, maar dat hierdie veranderings meer uitgesproke is in nasate van hipertensielyers, dat hulle heftiger is in persone wat 'n hiperreaksie tot die koue pressortoets toon en dat sekere indiwidue meer geneig is tot die vaskulêre gevolge van tabakrook. Dit is onprakties om elke ‚voornemende' roker ten opsigte van hierdie faktore te bestudeer, maar as ons die feite aan hulle bekend maak, het ons 'n deel van ons plig nagekom. Daar behoort ook gewys te word op die feit dat adverteerders van sigarette hierdie feite nie betwis nie, maar dit verswyg, net soos drankhandelaars nie ontken dat misbruik van drank tot dronkenskap en sy gevolge lei nie, maar dit verswyg met die wete dat die gevare van drankmisbruik baie goed bekend is onder die publiek.

Die siek persoon. Wat vaskulêre siektes betref, is ons vandag so goed bekend met die verwantskap van rook tot trombo-angiitis obliterans dat dit futiel is om enige vorm van behandeling te probeer toepas as die pasiënt nie ophou rook nie. Die siek vaatbed kan nie die vasokonstriksie van tabakrook ongestraf verduur nie en in 'n chirurgiese tydskrif is 'n paar jaar gelede gewaarsku teen die gebruik om sigarette aan gewondes te gee aangesien die vasokonstriksie kan lei tot die verlies van ledemate.[7]

Na aanleiding hiervan voel ons dat die geneesheer streng behoort op te tree met pasiënte met vaskulêre siekte en kroonaarsiekte, wat weier om die rookgewoonte te staak, omdat enige terapie onder sulke omstandighede gedoem is tot mislukking. Hierdie uitspraak is net so rasioneel soos 'n verbod op aktiewe sport kort na 'n miokardiale infarksie.

Volgens werk wat Doll, Avery-Jones en Pygott[8] onlangs gepubliseer het, is daar nie bewys dat die rook van sigarette peptiese ulsera veroorsaak nie, maar wel dat dit die genesing van ulsera vertraag en help om 'n chroniese toestand te veroorsaak. Hier kan ons die pasiënt dus waarsku dat sy rekening waarskynlik op die lang duur groter sal wees as hy nie sy rookgewoonte staak saam met die aanvang van sy ulserbehandeling nie.

Die werk van Clemmesen[9] en Lillienfeld[10] wat rook as etiologiese faktor van blaaspapilloom ter sprake bring, is tans nie beslissend nie, maar dui tog op 'n verdere faktor wat ons aandag moet geniet.

Kritiek op die stellings wat ons hier uitgespreek het, is vanselfsprekend. Mag dit dien om die aandag van die geneesheer by 'n netelige probleem te bepaal en laat ons by enige kritiek in gedagte hou die woorde van J. H. Robinson: ‚Die meeste van ons sogenaamde redenasies bestaan

daaruit om argumente te vind oor hoe om voort te gaan om te glo wat ons reeds glo.'

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THE RELIEF OF PAIN

Pain and its management constitute ancient reasons for the existence of the physician. The management of pain has at all times called for skill in diagnosis and treatment. It still remains a difficult clinical problem.[1] Among the great achievements of medicine in the last 25 years has been the alleviation of suffering and the removal of the cause of pain. Together with progress in many fields of medicine such as surgery, orthopaedics, radiation therapy, psychosomatic medicine and psychiatry, a great many pain-relieving remedies have been developed. These remedies include not only the various analgesics, but also sedatives of different types, placebos, local anaesthetics, and a heterogenous group of drugs effective in only some types of pain.

Pain is not easy to define—it is a highly personal experience, subjective in nature and complex as a physiological and psychological phenomenon. Scientists today conceive pain to have two component elements: the perception of pain and the reaction to pain. The total experience is composed of the sensation of pain and the associated sensations, emotional reactions and affective states.

Pain has certain characteristics, and one of these is its differing quality, according to the tissue structures involved. When superficial structures are involved, pain from noxious stimulation has a sharp quality, well localized, and quickly felt. The same stimulation applied to fascia, periosteum or tendon induces pain of a duller and more diffuse nature. In the viscera the pain is usually described as dull, aching, sickening, and difficult to localize.[1] Reactions to pain stimuli include other local tissue responses, neural, muscular, glandular, and the development of increased excitability within the central nervous system. Clinically these responses are represented by local tenderness, reflex skeletal muscle spasm, vasospasm, hyperalgesia and hyperaesthesia. Physiological responses such as these sometimes occur in anaesthetized or unconscious subjects. Certain of the responses may act as new sources of stimulation which may establish a vicious circle. The pain may outlast the original stimulation. It may become located away from the deep-tissue lesion or site of stimulation, as a 'referred pain'.

Another characteristic of pain is its intensity, which is rarely constant for any prolonged period. Mechanical, chemical, and environmental factors modify the intensity of pain. The severity of pain or the extent of suffering is independent of the size of the lesion, and although pain gives warning, it gives no true indication of the extent or the severity of tissue damage. There are other factors which determine the intensity of pain such as the significance of its origin, or debility, fatigue, anxiety and lack of sleep on the part of the sufferer.

In considering the methods available for the control of pain, it is necessary to assess the patient as a whole and to decide to what extent primarily structural, physiological, or psychological factors need attention. Psychiatric technic is important in managing patients with severe intractable pain. The main treatment of pain of a structural origin and from altered physiological function is concerned with the eradication of the primary disease. An imposing variety of drugs is available for this purpose, including antibiotics, antispasmodics, skeletal muscle relaxants, vasodilators and vasoconstrictors. Surgical removal of the lesion or physical methods of treatment may also be indicated.

There are many instances where the cause or mechanism of protracted pain is not easily evaluated. Under these circumstances much time and effort are needed, a detailed history should be taken and a thorough physical examination performed if optimal results are to be expected.

Consideration cannot be given here to a detailed discussion of the effects and complications of narcotic (addictive) analgesics. It is clear that these remedies should be avoided when other agents can be effective.[1] There is no addictive drug which is devoid of undesirable effects. The smallest possible dose that will produce the desired therapeutic effect is the best. While morphine is the classic analgesic, there are patients to whom this drug cannot be given. There are many other drugs that have their uses, both opiates (natural and synthetic alkaloids) and 'opioids' such as pethidine, methadone, and levorphan. Non-addictive analgesics are less potent and are chiefly used for the control of mild pain. The nature of their limited and selective action is unknown. What is urgently required is a more potent non-addictive centrally-acting analgesic than is available at present.

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‚GENEESKUNDE'

Op 31 Januarie 1959 het *Geneeskunde* in Pretoria verskyn. Hierdie tydskrif is 'n uitsluitlik Afrikaanse geneeskundige tydskrif onder die redaksie van prof. H. W. Snyman, bygestaan deur drs. O. V. S. Kok en W. J. Pepler, en uitgegee deur die firma Juta en Kie. Bpk., Posbus 1010, Johannesburg.

As mondstuk van die Mediese Vereniging van Suid-Afrika wil die *Suid-Afrikaanse Tydskrif vir Geneeskunde*, graag sy hartlike verwelkoming betuig aan hierdie nuwe Afrikaanse tydskrif.

Die doelstelling van *Geneeskunde*, soos uiteengesit in die redaksionele artikel, is om te voorsien in 'n steeds skerper gevoelde behoefte by studente aan geneeskundige artikels in Afrikaans—ook om ten dele te vergoed vir die gebrek aan Afrikaanse leerboeke. Daarby wil die tydskrif verteen-

woordigend wees van al die gebiede deur 'n fakulteit van geneeskunde bestryk, en daardeur die eenheid van begrip beklemtoon wat die moderne geneeskunde behels.

Ons wil hierdie nuwe tydskrif van ons volle medewerking ten alle tye verseker, veral ten opsigte van ons gesamentlike doelstellinge om Afrikaans as mediese vaktaal tot sy volle reg te laat kom en 'n eervolle plek te laat inneem in die ry van tale.

TOXOPLASMOSIS IN SOUTH AFRICA

1. THE PRESENT CONCEPT OF TOXOPLASMOSIS

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Toxoplasmosis has become established as an important parasitic disease of man and other warm-blooded vertebrates. Of world-wide distribution, it produces effects ranging from asymptomatic invasion to fatal infection. In contrast to surveys of incidence and case reports emanating from other countries, only a few isolated cases have been described in South Africa.[1-4] This may be associated with lack of clinical recognition, inadequate facilities for laboratory confirmation of infection, or an actual low rate of incidence. The purpose of the author is to try to aid the elucidation of these features by a brief survey of the present concept of the disease and the eventual establishment of adequate diagnostic facilities in South Africa and, in due course, to utilize these laboratory methods in a serological survey to gain an indication of the rate of incidence in this country.

I. THE PRESENT CONCEPT OF TOXOPLASMOSIS

Though reviews of the literature have accompanied cases reported in South Africa, an attempt is made in this article to acquaint the practitioner with the developments of more recent years in an outline of currently accepted views.

DISTRIBUTION

Geographic. Broadly, human infection appears related to geographic and climatic factors. Available information indicates a rather greater frequency in warm humid climates than in warm dry climates, whilst Icelanders show a low incidence and Alaskans are almost free of antibodies. Unfortunately, no large-scale population survey has been undertaken in tropical Africa; it would do much to substantiate or refute this opinion. Human cases have been reported from nearly all the countries of Europe, namely, England, Norway, Sweden, Finland, Holland, Belgium, France, Germany, Austria, Poland, Yugoslavia, Czechoslovakia, Hungary, Switzerland, Portugal and Italy. In Africa few cases are on record but reports have come from the Congo, Nigeria and the Union of South Africa. In the Americas cases have been described from the USA and the countries of South America; in the East from India, New Zealand, Tasmania, Australia, the Phillippines, and recently 5 cases from western Japan. The distribution amongst lower animals presents an equally wide geographical dispersion.

Species. The species distribution of the infection is unusually wide, for the causative parasite is without host specificity among warm-blooded vertebrates. Naturally occurring toxoplasmic infection has been proven in man, dogs, cats, wild rats, the gondi (a North African rodent), rabbits, hares, mice, rats, bandicoots, guinea-pigs, cattle, swine, sheep, silver foxes, squirrels, chinchillas, voles, kangaroos, wombats and wallabies. Amongst primates infection has been described in the baboon and chimpanzee. Many species of bird have been shown to harbour the parasite including pigeons, ducks, fowls, grouse and sparrows.

THE PARASITE

Toxoplasma gondii was first described in 1909, by Nicolle and Manceaux,[5] who observed the intracellular forms of the parasite in smears taken from the spleen and other organs of a North African rodent, *Ctenodactylus gondii.*

Classification

Satisfactory classification of *Toxoplasma gondii* has remained problematical. Originally, Nicolle and Manceaux[5] considered the parasite to resemble leishmania but recognized the absence of a kinetoplast. The characteristic lack of host specificity tends to set this organism widely apart from other protozoa, especially the sporozoa, though van Thiel[6] has suggested a striking similarity to the gregarines.

Thus, at present, *Toxoplasma gondii* may be considered a protozoan of doubtful systemic position, grouped with *Pneumocystis carinii* and sarcosystis.

Morphology

The classic morphology is best observed in the extracellular forms found in peritoneal exudate obtained from mice which have been experimentally infected by previous intraperitoneal inoculation with toxoplasma. Such exudate contains an average 300-400 million organisms per c.c. They fill extravasated leucocytes lying in the exudate, and great numbers of parasites lie free in the fluid, floating singly, clustered together, or recently liberated and packed around ruptured cells.

The organism is large, 3-7 μ long and 2-4 μ broad, crescentic in shape, and resembling a diminutive banana in outline, the one extremity being more pointed than the other, and the nucleus lying towards the blunt pole. A kinetoplast is not demonstrable. Staining with Wright's or Giemsa's stain reveals a light-blue cytoplasm and a red nucleus. Electron microscope studies of toxoplasma in thin sections of tissue culture by Meyer and Mendonca[7] indicate the absence of organs of locomotion. A thin periplast appears to surround the organism and becomes thickened at the two poles. At one pole the thickening resembles a ring to which long darkly staining internal structures are attached. At the opposite pole a distinct opening is apparent. Similar studies[8] of whole mounts and sections of toxoplasma indicate a large conoid at the anterior extremity measuring 1·3 μ, which can be invaginated into a collar and probably utilized to bore into the host cell. It is described as being accompanied by 14 fine fibrils extending fan-wise posteriorly. Phase-contrast microscopy of peritoneal exudate of infected mice[8] shows a similar pointed spicula at the anterior extremity used to penetrate host cells.

In tissue sections, fixation renders the toxoplasma shrunken and smaller. When acted upon by antibody they tend to become ovoid and less clearly defined. When a wet preparation is allowed to stand, the parasites enlarge and become more bulky, the cytoplasm often showing a vacuole and appearing granular.

The micro-organisms most likely to be confused with toxo-

plasma on a morphological basis are encephalitazoon, sarcocystis, leishmania, histoplasma and *Trypanosoma cruzi*.[9]

Physiology

Like viruses and rickettsiae, the organism is essentially an obligate intracellular parasite. It has thus never been cultivated in artificial media in the absence of living cells. The cellular situation of toxoplasms is always intracytoplasmic and no observation has been made of nuclear invasion. The lack of host specificity is further manifest at cellular level; the parasite will invade and multiply within any cell of the body except the red cell of the blood.

Reproduction was considered to be by longitudinal binary fission occurring repeatedly within the invaded cell. Weinman[9] describes a division rate of once every 6 hours during the first 24 hours. Recent investigation by Goldman *et al.*[61] clearly indicates that reproduction is in fact unusual in mode and by a process of internal budding they aptly term endodyogeny, in which the parent cell develops 2 daughter cells within it and finally disintegrates to liberate 2 toxoplasmic offspring. The invaded cell becomes packed with parasites, and paired parasites are frequently observed both within cells and without. Schizogony does not occur. Eventual rupture of the host cell liberates the contained parasites, which enter new cells, either phagocytic or non-phagocytic, to repeat their development.

Routine performance of frequent dye tests, from December 1956 to the present time, has necessitated continued passage of the parasite at intervals of a few days in mice and in simian kidney tissue cultures, to maintain the supply of viable parasites required for the tests. Associated with the passaging there is repeated microscopic observation of live parasites in infected mouse exudates and in tissue cultures. During this period I have not once observed the penetration and entry of a toxoplasm into a cell, nor have I seen reports of the actual moment of cell penetration.

Motility is exhibited by free extracellular organisms. It is best observed by the microscopic examination of wet cover-slip preparations on a warmed slide of infected mouse peritoneal exudate harvested and maintained at 37°C. Of a number of parasites clustered, apparently motionless, either clumped together or around a newly ruptured parisitized host cell, one parasite may drift away from time to time, pointed extremity foremost, with a slow momentum gradually falling away, to a distance of some 5 times its own length. After a momentary pause it tends to slowly return to its parent cluster. This action may be repeated several times. Eventually the parasite fails to return and slowly moves away as a lone voyager in the fluid. These free parasites show a definite rocking movement, which is barely perceptible, and a slow gliding movement easily confused with drift associated with currents beneath the cover-slip. Rolling of the crescentic parasite about its long axis, which may or may not be produced by the parasite itself, often suggests a rotary questing movement of the pointed extremity.

Preliminary observations on the metabolism of *Toxoplasma gondii* by Fulton and Spooner[10] have indicated the rate of consumption of oxygen and production of carbon dioxide. They have further studied the rate of utilization of glucose, the major substrate for respiration, and shown that respiration is inhibited by cyanide. Summers[11] has indicated that vitamin B6 is necessary for the growth of the organism.

In contrast to the proliferative form of *Toxoplasma gondii* thus far described, a pseudocystic phase of the parasite occurs in subclinical and chronic infections. In infections reaching these stages the toxoplasms may multiply within a number of cells which, however, fail to liberate the parasites by rupture, the nuclei becoming flattened and granular, and the cytoplasmic residuum reduced to a fine peripheral remnant forming a 'cyst wall'. The parasites themselves apparently have no part in the formation of this membrane. The pseudocyst may contain great numbers of parasites, which are maintained and protected in this situation from antibody and drug action. The experimental crushing of a pseudocyst will liberate the contained toxoplasma cells, each viable and capable of initiating a chain of cellular invasion in any suitable animal host, even after prolonged periods of dormancy within the pseudocyst. Pseudocysts may be observed in sections of any organ, but are particularly frequent in the brain. This phase of the parasite is undoubtedly of great epidemiological significance.

The proliferative form appears to have no unusual resistance to the external environment outside its host, to physical agents such as low heat or changes in osmotic pressure, or to the common antiseptics. Simitch *et al.*[12] report that toxoplasma survives no more than 10 minutes in water, but in saline survival may be prolonged to 24 hours, and that they will survive in smears made on glass slides and exposed to dessication for 3 hours at room temperature and 1 hour at 37°C. Jacobs, Jones and Melton[13] consider that handling of the parasite during the process of animal passage should be limited to 1 hour because, even in such a short time, deleterious effects on the toxoplasms may result from various suspending media used, with considerable loss of viability. They indicate that under the best circumstances survival in saline is limited to 2-3 hours, but that serum-saline is a superior suspending medium, the parasites being viable after 4-5 hours. My experience with the parasite is the same.[13] Lyophilization is unsuccessful. Prolonged storage at −70°C is impossible unless the temperature is slowly brought down to this level and the organisms suspended in glycerol. In tissues of animals dead of the infection, survival of the proliferative stage of the parasite must be of relatively limited duration. Simitch *et al.*[13] report that toxoplasms remained alive for over 2 days at room temperature and over 4 days at 4°C in carcases of sousliks (a squirrel species) dead of toxoplasmosis.

The pseudocyst form is more resistant and in infected tissue is undoubtedly mainly responsible for the recovery of viable parasites from such material after quite considerable periods have elapsed. This is especially so in chronic infections where the toxoplasms are present in the pseudocystic phase. Weinman and Chandler[14] accept that toxoplasma can survive for at least 10 days in pork refrigerated at temperatures of 2-5°C.

These are indications of the properties of resistance of the proliferative and pseudocystic forms. It is possible that they happen to be the only phase of a life cycle with which we are cognisant. Forms more resistant to the external environment may exist and should they exist, and be discovered, the unknown mode of transmission of this challenging parasite may be unveiled.

Weinman[9] has described a potent factor associated with the organism and termed it toxotoxin. It is produced *in vivo* and occurs in the ascitic fluid of intraperitoneally infected animals. Campbell, Mackay and Vantsis,[15] working on canine toxoplasmosis and the isolation of toxoplasma from a dog, confirmed the presence of this very toxic substance in the peritoneal fluids of infected mice and of an infected dog. However, other workers have failed to find evidence of any such substance and Cook and Jacobs[42] were unable to demonstrate the production of this factor in toxoplasma-infected tissue cultures.

Variation

The RH strain of *Toxoplasma gondii* was isolated by Sabin from the brain of a fatal case of encephalitis occurring in a 6-year-old boy in 1939. It bears the initials of this luckless child and was considered to have originated from a cat in the household which died from an unknown cause before the child became ill. This strain is maintained and traditionally used by all laboratories performing the dye test. To retain comparability of dye-test results from different countries it has been necessary to use an identical strain of parasite—the RH strain, a regrettable necessity since it is of high virulence.

The CH strain was isolated by Dr. H. R. Siebold of the US Department of Agriculture from chinchillas dying of a toxoplasmic epidemic on a farm in New Jersey.

Strain 113 originated in the laboratory of Dr. Leon Jacobs in the USA. It is of particular interest as a strain of decreased virulence resulting from passage of the RH strain through the larval and nymphal stages of the tick *Rhipicephalus sanguineus*.

Since these earlier isolations toxoplasms have been isolated from a wide variety of mammals from many parts of the world. The major difference between all these strains is a variation in virulence, depending on the host of origin. The essential feature is that all known strains of toxoplasm

are morphologically identical and, with practically no doubt at all, of a single species—*Toxoplasma gondii*.

Culture and Maintenance

This sensitive organism, requiring live-cell maintenance, may be cultured in the laboratory animal, in tissue culture, and in the developing chick embryo. It may further be maintained by prolonged storage at −70°C when suspended in glycerol, or in chronically infected rats.

Laboratory Animals. White mice are most satisfactory for this purpose. They are very susceptible to infection with toxoplasms and any route of inoculation is successful—intracerebral, intraperitoneal or subcutaneous injection, intranasal instillation, deposition on intact mucous membranes and feeding on infected tissue containing pseudocyst forms. In practice, intraperitoneal inoculation is easy and is the method generally used for maintenance of strains. The plentiful peritoneal exudate formed gives a rich harvest of toxoplasma, the fluid being used for the regular 4-5 day passage of the strain from one batch of mice to another, and for the performance of the dye test. With the RH strain in laboratory use, the fluid is mainly devoid of parasites for the first day after inoculation; a few become manifest 24 hours later, rapidly increasing to very great numbers up to 48 hours, and thereafter declining in number. The strain is uniformly fatal to mice some 5-8 days after inoculation.

Tissue Culture.[16-19, 42] The growth of *Toxoplasma gondii* is well and rapidly adapted to mammalian cell cultures, there being progressive invasion of cells, which are disrupted to release free organisms into the fluid nutrient medium. After long periods of tissue culture the parasite retains its virulence as shown by mouse inoculation. Tissue cultures inoculated with toxoplasma and incubated at 37°C show the highest number of parasites 5-7 days later. The method provides a satisfactory source of antigen for the complement-fixation test, and infected simian kidney tissue cultures are used for this purpose in our laboratories. We have maintained the parasite by routine mouse passage and simultaneously in simian kidney tissue culture to ensure that any mishap in one or other method of culture will be less likely to result in loss of the strain. In the author's experience tissue culture has not proved to be a satisfactory source of toxoplasma for the dye test because, prolific though development may be, it does not produce enough organisms and too many non-viable forms are present.

Developing Chick Embryo.[19] The introduction of toxoplasma into the chorio-allantoic, amniotic or yolk sacs of the 6-12-day-old developing chick embryo will almost invariably result in embryonic death 8 days later. Large yellow nodules are found on the allantoic membrane and scattered lesions in the haemorrhagic embryo. Toxoplasmas are numerous in these sites. Antigen for the complement-fixation test was originally, and is most usually, prepared from infected embryonated eggs.

Prolonged Storage at −70°C.[20-21] This procedure has been a notable advance toward obviating the time-consuming and rather dangerous necessity of repeated animal inoculation in order to maintain a given strain. A suspension of the parasite, containing 5-10% glycerol, is refrigerated and then frozen in a bath of dry ice in alcohol at −15°C, and the temperature is then slowly dropped to −70°C. The parasites are maintained at this temperature in the deep freeze. The salient factors of success in this method are the slow dropping of the temperature and the use of glycerol. Parasites may be stored very successfully for 6 months under these conditions, when an inoculation made from the thawed suspension into laboratory mice will produce virulent infection. Chandler and Weinman[20] reported the successful storage of toxoplasma for 184 days by this method. Eyles, Coleman and Cavanaugh[21] emphasize that, at present, the best methods preserve only a small percentage of the toxoplasma exposed to freezing, but have demonstrated survival up to 209 days. In one of our experiments a suspension which had been stored by this method for 270 days was thawed. Microscopic examination revealed morphologically intact toxoplasma but intraperitoneal inoculation of mice with the material failed to produce infection. These mice were challenged 6 weeks later with a small inoculation of virulent organisms, in the hope that the apparently non-virulent toxoplasma would have stimulated the production of protective antibody. All the mice rapidly succumbed. Ordinary freezing or the use of glycerine instead of glycerol is unsuccessful. Roger and Roger[22] have tried slow freezing in a saccharosed isotonic solution, followed by rapid thawing by immersion of the containing ampoule in boiling water. They showed that infectivity for mice though decreased, was thus maintained for 1 month.

Chronically Infected Rats. Parasites may be maintained in pseudocysts in the brains of chronically infected rats. Rats are relatively resistant and are given non-lethal inocula of toxoplasma. When required, an emulsion of infected rat brain is used as an inoculum for experimental animals. Van Thiel[23] working with albino rats and 4 Netherlands strains of toxoplasma maintained 1 strain for a year in this manner and the other 3 strains for a month. The method has little advantage with the advent of prolonged storage at −70°C.

HUMAN INFECTION

There is no significant difference in the apparent incidence of this infection between the human sexes.

Age groups have shown repeatedly a classic distribution of incidence in serological surveys, the number of positive reactions being low in the first decade of life and increasing with age. A characteristic example is the findings of Feldman[24] who, using the dye test and a positive reaction in a titre of 1 : 16 as significant, conducted a population survey in Pittsburg, Pennsylvania. His findings are summarized in the following table.

Age in years	*Percentage Positives*
0— 4..	5
5— 9..	10
10—29..	25—30
30—39..	45
40—60..	65—70

The incidence of human infection is exemplified by publications such as that of Feldman and Miller[25] on the serological study of toxoplasmosis prevalence, who, in addition to 24 animal populations, investigated 10 geographically different human populations. Positive dye-test percentages in human groups varied from 0% in Eskimos to 68% in Tahitians. The only published serological indication available to date of possible human prevalence in South Africa is the results obtained by Schneider, Goddard and Heinz,[26] who used the toxoplasmin skin test in a survey of Africans in Johannesburg and demonstrated the usual increase of positive reactions with increasing age and a total incidence of 31%.

It is now very evident that there is no reason to doubt the specificity of serological diagnostic tests in current use, a matter to be discussed below. Accepting these findings it might be concluded that toxoplasmosis is one of the commonest infections of man. This conclusion has long been considered unjustifiable, but it may in fact be within the bounds of truth. It must, however, be understood that there may be much infection with little clinically manifest disease; toxoplasmosis would appear to be a long-established infection of warm-blooded vertebrates with a good host-parasite relationship.

Toxoplasmosis has been known as a disease of man since 1939 and is now being recognized with greatly increasing frequency. Human infection may conveniently be elaborated from the following classification: (1) Congenital, (2) acquired —(*a*) sub-clinical, (*b*) glandular, (*c*) ocular, (*d*) encephalitic, (*e*) exanthematous.

Congenital

Congenital infection was the first form of the disease to be described and hundreds of cases have been diagnosed in various parts of the world. Evidence shows that a foetus becomes infected only if the mother acquires her first toxo-

plasmic infection during the associated pregnancy. The infection in the mother is generally entirely subclinical. The author has not been able to find any report of a woman giving birth to more than one toxoplasmic infant. Wright[27] states that in the entire world literature there is no authenticated instance of a mother giving birth to a toxoplasmic child in any subsequent pregnancy, and that there is no record of a woman with antibody at the start of pregnancy producing an infected child. Millions of women with toxoplasmic antibody give birth to children without toxoplasmosis.[28] Feldman,[29] in a study of 103 congenital cases, reiterates that at present it appears that congenital infection is only a rare manifestation of a commonplace disease.

If the foetus becomes infected during the early stages of a pregnancy, the central nervous system and the eyes show the greatest evidence of parasitic activity, as antibody elaboration causes the parasite to regress from the viscera to the partial protection from antibody action afforded in these two situations. Infection during the latter part of pregnancy affords insufficient time for the elaboration of sufficient antibody antagonism to the parasite, with resultant symptoms indicative of extraneural visceral parasitic activity in addition to that in the nervous system. Thus clinical manifestation may be very varied and the original classical tetrad of symptoms described, namely, hydrocephaly or microcephaly, intracranial calcification, convulsions, and chorioretinitis, is demonstrated in a small proportion of cases only. This is supported by 150 cases studied by Eichenwald,[30] whose findings suggest that toxoplasmosis should be considered as a possible diagnosis of almost any obscure illness during the neonatal period.

Broadly, then, before parasitic regression to the brain, any or all of the following may be demonstrable: Fever, jaundice, a maculopapular rash, splenomegaly, hepatomegaly, pneumonitis, myocarditis, and hydrocephaly or less frequently microcephaly. When the infection has occurred earlier in pregnancy the clinical picture described tends to merge into the more classical symptomatology of convulsions, hydrocephaly or more rarely microcephaly, chorioretinopathy and radiological evidence of cerebral calcification. In survival to infancy psychomotor retardation is usually clearly present. Mild cases of foetal infection may occur.[31] Chronic signs of infection in apparently normal infants may show as reactivation of chorioretinopathy later in life due to activity of surviving ocular pseudocystic parasitic forms.

Eichenwald,[30] in his study of 150 congenital cases, followed most of them from birth to 5 years of age, and found a mortality of 12%, whilst 90% of survivors showed mental retardation, 75% developed convulsions, spasticity and palsies, and about half of them had severely impaired sight. The condition may closely simulate haemolytic disease of the newborn. Attention is drawn to this point by Bain *et al.*[32] in reporting 3 illustrative cases. Foetal death with abortion and stillbirths may be associated with the intrauterine parasitic invasion and toxoplasma has been isolated from products of conception in such cases.

Cerebrospinal fluid and blood pictures are not specific and are rarely of assistance in diagnosis. In the cerebrospinal fluid xanthochromia may be present, protein possibly increased, sugar levels often decreased, a pleocytosis not infrequently present, and erythrocytes sometimes noted. In the blood eosinophilia may or may not be present.

It is apparent that the varied symptomatology makes a clinical diagnosis inordinately difficult. Essentially, as in all forms of the disease, clinical suspicion requires laboratory confirmation for a final diagnostic evaluation.

Acquired

Congenital toxoplasmosis, being the first type described, has received the major share of recognition, but the significance of the acquired disease forms is increasingly apparent.

Subclinical. The high incidence of antibodies in the general population, coupled with general inability to elicit any clear-cut history of toxoplasmic illness from members of the general population presenting antibodies, or from mothers of toxoplasmic infants, suggests that subclinical infection is the most frequent form of invasion in man. However, it is not improbable that clinical features may be manifest which, thus far, are not thought to be associated with the aetiological agent.

Glandular.[33-36] The lymphoglandular form is a frequent manifestation of acquired infection. There is no special age or sex incidence. It may run a febrile or an afebrile course. If afebrile the patients often accidently discover the enlarged glands themselves, though not suffering any other signs of illness at the time. It is likely that many unnoticed infections of the so-called subclinical type are, in fact, cases where lymphadenopathy is the only presenting sign but escapes detection. During the early stages of the infection there is an initial parasitaemia. In acute febrile cases the fever generally persists about 2-3 weeks. The associated lymphadenopathy frequently persists for 6 months and longer. The whole course of the illness is protracted, but usually benign, with complete recovery without complications. Severe fatigue is a very marked feature of most cases and lasts for many months during the convalescent phase. The enlarged lymph glands, after initial tenderness, are painless, firm and discrete, with no unusual attachment to surrounding structures. Involvement may be of one area only, such as the neck, or relatively generalized. The spleen is seldom palpable. A prominent finding in the blood in most cases is a relative lymphocytosis, with atypical large lymphocytes resembling those found in glandular fever. The Paul-Bunnell test is negative. Thus, any case presenting with the physical signs of glandular fever and supportive evidence in the blood findings, but with a negative Paul-Bunnell test, must arouse suspicion of toxoplasmosis. In such cases sero-reactions for toxoplasmosis should be tested. Positive reactions in rising or very high titre will confirm the suspected diagnosis and an attempt should be made to isolate the parasite from lymph-node biopsy.

Ocular. Attention was drawn to the existence of acquired toxoplasmic ophthalmitis by reports such as those of Wilder,[37] who studied a series of 53 enucleated human eyes presenting granulomatous uveitis. She identified toxoplasma morphologically in sections, and this was substantiated by a serological follow-up of her patients. The parasite was then successfully isolated from enucleated eyes; Jacobs *et al.*,[38] identified toxoplasma cultured from the macerated inner layers of an adult human eye enucleated for painful blindness due to chorioretinitis of 8½ years' duration, and Pillat and Thalhammer[39] from a case of iridocyclitis which was the sole manifestation of acquired toxoplasmosis. Jacobs *et al.*[40] found that 35% of 298 cases of granulomatous uveitis were apparently toxoplasmic in origin. Choroiditis has been

described in association with the glandular form of the disease. However, the majority of toxoplasmic infections pass unrecognized and are benign; small residual choroido-retinal lesions are noticed because symptoms will readily be produced in this situation. The eye condition may arise as a delayed manifestation of infection acquired long before.

Encephalitic.[3, 27] This form of the disease is described as acute with a sudden onset often following a period of some months of feeling vaguely unwell. Manifestations are pyrexia, stupor, papilloedema and unconsciousness. Concomitant signs of lymphadenopathy may be present. A fatal outcome is usual. The cerebrospinal fluid is described as under increased pressure with a pleocytosis of lymphocytes and polymorphonuclear leucocytes. Wright states that only 7 cases are described in the medical literature. Nevertheless, serological evidence has indicated that 3 cases of benign meningo-encephalitis observed in Johannesburg during the course of this study were due to toxoplasmic infection. A brief review of a preliminary study of the dye test and of these cases will be published shortly.

Exanthematous.[33] The infection closely resembles typhus fever clinically. It is acute, with sudden onset accompanied by chills, fever and a maculo-papular rash which does not involve the scalp, palms and soles. Pneumonic symptoms are frequent and myocarditis and meningo-encephalitis may be present. It usually ends fatally. This form of the disease also appears to be rare.

In general, clinical presentation may differ within wide limits depending on the anatomical site most involved. Pulmonary forms closely resembling pulmonary tuberculosis are reported. It may present as a space-occupying lesion of the brain. Accidental laboratory infections[43] have been known to confirm the clinical findings.

Toxoplasmic infection as an aetiological factor in conditions such as schizophrenia and epilepsy should always be considered.

Pathology of Human Infection.[3, 44-46] In the glandular form lymph-node biopsy shows characteristic features. The normal structure of the gland is present but there is cortical hyperplasia with obvious follicles which show pale cells with large darkly-staining nuclei. Abnormal nuclei are not evident but mitotic figures are frequent. Macrophages are plentiful and contain blackish granules, which are also found outside the cells. Intact toxoplasma are very rarely seen in these sections.

Pseudocysts packed with toxoplasma are often seen in sections of the brain and eye and may be found in subjects coming to autopsy for some unrelated cause. A striking feature is the absence of local reaction around these large structures.

The acquired type with cerebral involvement shows a necrotizing demyelinating encephalitis with gelatinous softening. Free toxoplasma or pseudocysts may be seen on section of the brain or other organs.

In congenital cases the brain shows internal hydrocephalus (rarely microcephaly) with associated aqueduct stenosis and arachnoiditis with cellular infiltration of plasma cells, mononuclears and lymphocytes. The free form of the parasite is often seen and pseudocysts are usually present. In other organs similar reactions may be featured.

Diagnosis. The variability of the symptoms makes certain clinical diagnosis improbable, and laboratory confirmation is essential.

Laboratory methods of diagnosis are (1) isolation of the parasite, (2) the Sabin-Feldman dye test, (3) the complement-fixation test, and (4) the skin test.

1. *Isolation of the Parasite*

The most certain way of proving the infection is to demonstrate the presence of the parasite, but in the past this has been difficult to achieve. However, with improved understanding of the physiology of toxoplasma very much better results are being obtained. A successful method is by animal inoculation; white mice are most satisfactory and have the added advantage that laboratory strains do not appear to be subject to natural toxoplasmic infection. The essentials to possible success are as follows:

(*a*) Rapid transference of material from the patient to the experimental animal. The organism cannot survive long outside the host, and every moment lost is important. The material is ground and suspended in sterile serum saline and inoculated into mice treated with cortisone. Several subsequent passages are performed before the run is discarded as unsuccessful. If toxoplasma-like forms are isolated cross-immunological studies will finally prove the species should confusion arise from the presence of other similar organisms such as encephalitazoon encountered in the mice.

(*b*) The use of the proper body fluids or tissues from the patient:

(i) Material from a lymph node gives a good possibility of successful isolation especially in acquired infections.
(ii) The cerebrospinal fluid is most satisfactory in active congenital cases but tends to give very poor results in acquired infections.
(iii) The blood may give evidence of parasitaemia on inoculation into mice, but probably only within the first week or two after infection. This procedure is therefore seldom of use to the clinician who, except in very rare instances such as laboratory infections caused, for example, by the prick of an infected needle, will only see the patient after symptoms have developed and parasitaemia regressed.
(iv) Tissue obtained at autopsy, especially from the brain, heart, spleen, or lymph nodes, is valuable but again rapidity of transfer is the essence of success.

The demonstration of toxoplasma-like structures in stained films or in histological sections is most significant but the diagnosis is better confirmed by isolation of the parasite or serological investigation of the patient.

2. *Sabin-Feldman Dye Test*[47-52]

The reaction as described by Sabin and Feldman in 1948, though slightly modified by some workers, has firmly withstood the test of a decade and remains the corner stone of diagnostic procedure. The high incidence of antibodies in the general population, coupled with little active disease, indicated either an excellent host-parasite relationship or that the serological procedure was not reliably specific. It was reported that sarcosporidiosis, South American trypanosomiasis and trichomoniasis could all give falsely positive reactions with the dye test for toxoplasmosis. Sarcosporidiosis is excessively rare in man, and Chaga's disease, caused by *Trypanosoma cruzi*, occurs in a single geographical region. *Trichomonas vaginalis*, however, being so ubiquitous a parasite, would be of definite significance. However, more recent investigation by various workers has shown that,

apart from extremely rare cases of human sarcosporidiosis, the dye test is highly specific.

Basically, the test is conducted by heating the serum to be investigated for the presence of toxoplasma antibody to 56°C for 30 minutes to destroy any non-specific antitoxoplasmic factor, which may be present in some sera. Serial fourfold dilutions of the serum are then prepared in saline and to each is added a fixed proportion of toxoplasma suspension. This suspension consists of viable toxoplasma in a freshly-harvested highly-infected mouse exudate with a small amount of heparin to avoid clotting to which is added the essential accessory factor, considered to be associated with the properdin system, and used in the form of a non-immune human serum, which must be entirely free of toxoplasmic antibody.

The various serum dilutions with the added toxoplasma suspension are now placed in a waterbath at 37°C for 1 hour. If antibody is present in the serum it will, during this time, and in the presence of accessory factor, have an effect on the cytoplasm of the toxoplasma. This cytoplasm-modifying effect is made more easily recognizable by the addition of alcoholic methylene blue precisely buffered to pH 11. Therefore, after removal from the water-bath, fixed volumes of freshly prepared dye are added to each of the dilutions, which are then stored in the refrigerator for a short while. If adequate antibody is present in a tube the cytoplasm of most of the extracellular parasites in that tube will be so modified that it will not stain with the dye. Intracellular parasites cannot be reached by antibody, and so are stained in all cases. If antibody is not present the cytoplasm of the toxoplasma takes up the dye in a normal fashion. Any dilution of the serum is said to be positive if more than 50% of extracellular parasites are unstained when examined under the high dry power of the microscope.

With each batch of tests, known positive and negative control sera are investigated concurrently and a control is prepared to check the presence of sufficient viable parasites in the toxoplasma suspension. The lowest titre considered to be of diagnostic significance is 1 : 16.

3. *Complement-fixation Test*

The specificity of the complement-fixation test for toxoplasmosis is without doubt. Antigen for the reaction is usually prepared from the harvest obtained from the chorioallantoic membrane of the infected chick embryo, which gives a good yield without the difficulty of anti-complementary properties associated with antigen culled from infected mouse peritoneal exudate. In these laboratories antigen prepared from infected simian kidney-tissue cultures has been used extensively. Technically the test has the very great advantage that live toxoplasma are not used and that the antigen is stable and can be stored for long periods in the frozen or lyophilized state.

Interpretation of Results of Dye and Complement-fixation Tests

The clinical diagnosis of toxoplasmosis cannot be entertained without supportive serological findings in one or both of these tests. For purposes of diagnosis these two reactions must always be used in conjunction. The test of basic importance is the dye test, which must be used for all routine purposes. The complement-fixation test is auxiliary to the dye test and assists in detecting acute and more recently acquired infections. Complement-fixing antibody appears later and disappears earlier than dye test antibody; thus a negative complement-fixation test does not rule out toxoplasmosis, and the test cannot be used alone for the diagnosis of toxoplasmosis—a fact to be emphasized.

(*a*) *Acquired Infections.* In both clinically manifest and entirely subclinical infections, the dye test becomes positive 1-3 weeks after infection. The titre rapidly rises within a few weeks to 1 : 256 or even 1 : 16,384 and higher. Serial samples of blood must therefore be submitted to demonstrate a markedly rising titre, which is of the greatest significance. Further, a negative result followed by the development of a positive reaction and a rising titre in subsequent specimens is diagnostic. Titres in active infections usually reach high levels. The presence of a large percentage of positive reactions in the general population must be borne in mind and a positive dye test, especially in low and static titre, in no way indicates that a patient's present symptoms can of necessity be attributed to toxoplasmosis; but they may result from past infection.

Complement-fixing antibody usually develops more slowly and the reaction becomes positive about the 28th day after infection. The complement-fixation test is therefore negative at the time when the infection is most active and then becomes positive in rising titre.

Thus in acute infections of recent onset dye-test antibody is strongly present and the complement-fixation test negative during the early stages of the infection. The complement-fixation test then becomes positive and, together with the dye test, remains so in increasing titre during the following weeks. Both may persist in relatively high titre for a year or more. Complement-fixing antibody practically disappears after 6 years whilst the dye-test antibody slowly falls to persist in low titre, probably for life.

In infection acquired many years previously the dye test will be positive and the complement-fixation test negative. The dye test remains positive in low titre and the complement-fixation test remains negative.

(*b*) *Congenital Infection.* Maternal toxoplasmic antibody will pass the placental barrier. If the mother was infected before the associated pregnancy the antibody developed will be passed to the foetus but, as the parasites are adequately controlled in the mother by her antibody development, the baby will not be infected. The infant will, however, have a positive dye test due to the presence of maternal antibody. Such a positive finding in the newborn does not indicate congenital infection, and in these cases the baby's dye-test titre will slowly fall and be negative by about the 4th month after birth. The mother's dye test will be positive in a titre depending on the time elapsed since her infection, which period can be more accurately indicated by concomitant results obtained with the complement-fixation test. Even though the mother should acquire her primary infection during her pregnancy the foetus will not necessarily be infected. If, as a result of such infection, congenital toxoplasmosis of the baby ensues, the mother will have dye and complement-fixation tests positive in high titre and the baby a very strongly positive dye test and a complement-fixation test perhaps at first negative and then becoming positive in rising titre. These findings would indicate recent and active infection of the infant following on maternal infection acquired during pregnancy.

4. *Skin Test*

The reaction is a dermal sensitivity test and a positive reaction of the delayed tuberculin type develops in persons with circulating specific antibody. The antigen injected is the same used for the complement-fixation test. The test has the following disadvantages:

(*a*) The degree of local reaction does not indicate the amount of serum antibody.

(*b*) Some persons with positive dye tests will give a negative

skin test, and therefore a negative reaction does not indicate a definite absence of infection.

(*c*) There is considerable delay between infection and the development of a positive skin test.

The skin test consequently has no place in the diagnosis of an individual case, and is preferably reserved as a crude tool for investigating the incidence of past infection in population surveys.

Animal and roller-tube protection tests have little place now in serological diagnosis, but work on a possible haemagglutination test for toxoplasmosis[53] shows much promise.

Treatment[27]

Many substances have been investigated as possible therapeutic agents and discarded as being of little or no value. However, the antimalarial compound pyrimethamine (daraprim) in combination with one of the sulpha drugs, preferably sulphadiazine has given very good results indeed, the pyrimethamine and sulphadiazine having a marked synergistic effect one on the other. In addition to clinical and experimental experience in man and animal, the degree of protection afforded *in vitro* in tissue cultures of the parasite has been clearly demonstrated by Cook and Jacobs,[54] who have also shown that cells in such cultures can store pyrimethamine.[55] From the nature of the physiology of the parasite it may be assumed that the proliferative forms will be most susceptible to drug action whilst the pseudocystic phase is protected in variable degree, not only from antibody action, but from drugs as well. This, in fact, is the case. The acute stages of the disease show much greater response than more chronic infections, though chronic eye manifestations show a considerable degree of improvement or, at least, a usual halting of further deterioration, when treated with pyrimethamine and sulphadiazine.

In a considerable proportion of acute cases treatment with this drug combination results in cure and in other acute cases the effect is variable, the degree of benefit depending on the promptitude with which treatment is begun after infection.

Little improvement can be expected in congenital cases of any severity, since trauma occasioned by the parasite has reached an advanced stage of irreversibility. In less severe involvement, if symptoms do not regress, at least the development of later symptoms may be controlled.

A pregnant woman who develops positive serological reactions during the course of pregnancy should be offered the opportunity of treatment for possible protection of her baby. Her medical attendant must weigh his decision whether to treat her or not, considering the toxicity of pyrimethamine and the available knowledge of toxoplasmosis. However, it is desirable that pregnant women should have toxoplasmic serological investigations performed at the commencement of each trimester of pregnancy.

A dosage schedule recommended by several workers for an adult patient consists of a loading dose of 6 to 8 g. of sulphadiazine and 75 mg. of pyrimethamine, followed by a maintenance dose of 4 to 6 g. of sulphadiazine and 25 mg. of pyrimethamine daily for 14 days. An important disadvantage of therapy is the toxicity of pyrimethamine which, in adequate and prolonged dosage, may have serious untoward effects such as thrombocytopenia, leucopenia, megaloblastic anaemia or agranulocytosis, and close observation of haematological findings has to be maintained throughout treatment. The administration of 5 mg. of folinic acid daily is reported to diminish this toxicity and possibly allow the continuation of treatment without the withdrawal of pyrimethamine.[57]

Epidemiology

The epidemiological aspect of toxoplasmosis has been investigated in many parts of the world but, apart from direct infection in laboratory accidents, the method of transmission of the parasite remains unknown. Toxoplasmas have been isolated from a wide variety of animals and birds, including nearly all the domestic species, and in very many cases from apparently healthy hosts with no signs of active disease. The disease occurs in both enzootic and epizootic forms, as for example in the hares of Denmark, but so far no epidemics have occurred in man. It would appear that man is one of many usual vertebrate hosts for *Toxoplasma gondii*.

Repeated endeavours to demonstrate arthropod transmission as a significant mode of spread are completely unconvincing. It is unusual for healthy animals closely confined over long periods with successive batches of infected animals to show any signs of infection. For a better understanding of the unsolved epidemiological problem it is significant that the parasites are widely distributed through the bodies of infected hosts, and have been found in the parotid glands, sputum, intestinal contents, kidneys and milk of infected mice, in the saliva of rabbits before death, in the urine and intestines of dogs, lining intestinal ulcers in cats, in the eye exudate of infected dead pigeons, alive in pork which had been frozen for 10 days, in the saliva of a human case, and in the macrophages from the bronchioles and alveoli of a man with acute toxoplasmic pneumonitis. A history of the habit of ingesting raw eggs is on occasion associated with the onset of the disease in man. These are indications that the modes of transmission might be numerous.

It is possible that intimate contact with animals acutely ill of the disease may allow transfer of the parasite in secretions and excreta. As the proliferative form is probably destroyed to a large extent by the acid barrier of the stomach, entry is probably effected through the conjunctiva, mucous membranes or skin, especially where there is a breach of continuity of the surface. Infection might also occur during the preparation of carcases of infected hares or other animals for the table. Pseudocyst forms, being much more resistant, are more likely to pass successfully the acid barrier of the stomach, and experimentally, and very likely in nature, are more prone to give rise to infection by ingestion than the sensitive free proliferative form of the parasite.

The question is too vast to discuss at length but, in conclusion, a further indication of the nature of the problem is afforded by a case reported by Gibson and Eyles.[57] They investigated a newborn Negro infant dead of congenital toxoplasmosis in Memphis, Tennessee. Serological tests suggested that other members of the baby's family had recently been exposed to a common source of infection. Various stray animals were captured in the immediate environment and frequenting a garbage dump near the house of the family. The authors actually succeeded in isolating *Toxoplasma gondii* from 7 of 35 cats, 2 of 3 ducks, 3 of 7 chickens, 1 of 16 pigeons, 1 dog collected 4 blocks away, and 7 of 121 mice (5 of them amongst 20 caught in the family's house).

SUMMARY

1. A broad outline is given of the distribution, classification, morphology, physiology, variation and culture of the parasite *Toxoplasma gondii*.

2. Human infection and its forms of presentation, diagnosis and treatment are discussed.

3. Attention is drawn to the incomplete understanding of the epidemiological features of toxoplasmosis.

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A PROGRAMME FOR THE CARE OF CRIPPLES IN SOUTH AFRICA

G. T. DU TOIT, F.R.C.S. (ENG.), *Chairman, National Council for the Care of Cripples in South Africa, Johannesburg*

In considering a comprehensive programme for the care of cripples in this country one is limited to some extent by the existence of a multiplicity of organizations and Government departments which have acquired a vested interest in those aspects of the service for which they are responsible. Such organizations, having justified their existence by filling a need where no previous service existed, may resist attempts at amendment of their functions or re-definition of their limitations.

In order to get a comprehensive plan adopted and to work towards it in an organized manner it will be necessary to influence heads of departments as well as to modify the official policy of the Government in relation to field work, centralization, transport, hospitalization, after-care education, vocational training and placement of cripples. In countries like Sweden, Denmark, Holland, Switzerland and Austria considerable unanimity of opinion has been achieved in this type of work. In a country like the United States of America factors such as the high standard of living, the enormous resources, the alert public conscience of the well-to-do and the peculiarity of tax exemptions for charitable donations, all contribute towards the development of good services in many States without necessarily achieving uniformity.

In the Welfare State of Great Britain a most comprehensive programme for the care of cripples has been carried out, but the administrative authorities have shown great inertia. The general standard of care has been raised, but I believe that the personal relationship of patient to doctor, social worker and therapist has to some extent been lost in the levelling-off processes of a mass-production machine.

In this country, handicapped as we are by a dearth of skilled persons and a low economic status of a large proportion of the community, we are a long way from defining our problem, let alone solving it.

The following is a full statement of our needs for the present as well as for the future:

Orthopaedic Centres

Primary orthopaedic centres should be established in large cities attached to medical schools and teaching hospitals. A University orthopaedic and traumatic centre should have a director of the regional service, a number of orthopaedic specialists graded from the most senior consulting surgeon to part-time surgeons, full-time orthopaedic surgeons serving apprenticeship before going into practice or surgical registrars accumulating experience for registration purposes as required by the South African Medical and Dental Council, and house surgeons with surgical ambitions. A unit such as this should have a full complement of orthopaedic sisters, theatre nurses, plaster-room attendants, clinical secretaries, physiotherapists, occupational therapists, remedial gymnasts, orthopaedic mechanics, limb fitters, surgical bootmakers, etc. The unit should also have its own radiological department under a radiologist interested in this aspect of surgery and pathology, and there should be close cooperation with other departments of the medical school and teaching hospital.

For example, assistance from the departments of pathology and bacteriology would make possible more accurate diagnosis, more precise evaluations and more definite attacks on particular problems. The advice and assistance of the department of medicine would be needed in such problems as the lung-function assessment of paralytic polio cases and of scoliotic deformities of the spine, and fitness for operation. This department would also be able to assist in the alternative evaluation of results achieved—a guard against continuing outmoded and useless techniques—and a guide for future practice. The help of the department of thoracic surgery and the ear, nose and throat department would be useful in paralytic cases and in cases of deformity of the spine and chest.

The department of physical medicine should become more aware of the extent to which it can contribute to the welfare of cripples. The department of general surgery should integrate its experience of handling major and minor trauma, blood and fluid loss, major burns, as well as its experience in the many fields in which there is no real boundary between orthopaedics and general surgery.

The neurosurgical department has much to teach the orthopaedic surgeons in the fields of technique and diagnosis. Simi-

larly the orthopaedic surgeon could play his part in the rehabilitation, splinting and re-education of the neurosurgical case. Co-ordinated team-operating would not only raise the standard of work for a particular patient, but would provide invaluable training for the men in both departments.

Cases for amputation should be dealt with by an orthopaedic surgeon with special experience in the construction of artificial limbs. Today, not only does the mechanic build an artificial limb to fit the amputation stump, but the surgeon devises a stump which can be fitted with the most desirable type of limb. Artificial limbs and orthopaedic appliances manufactured in this country are still primitive, and the administration of this service is poor and under divided control.

Our Government departments do not yet appreciate the standard to which they should develop this science in the interest of the patient. Taking into consideration that polio may be progressively eliminated in a few years and that surgical tuberculosis will decline here, as in more advanced countries, we should prepare for the increase in the incidence of traumatic disabilities. Accidents are taking so great a toll of our skilled workmen, our youth and our best brains that the country is losing at least £1,000,000 of national wealth per year.

An integral part of the services rendered by the orthopaedic and traumatic department should be the training of medical and para-medical personnel. The social services required should include the contact during the hospitalization period, the follow-up at home and rehabilitation. Moreover, the vocational training for those persons who are permanently incapacitated needs special planning.

Regional Services

A large unit such as described would serve a large local population and long-stay cases and complicated cases over an area possibly hundreds of miles in diameter. For example, a primary unit in Pretoria would serve the North-Eastern, North-Western and Northern Transvaal. The Johannesburg unit would serve the South-Eastern, South-Western and Southern Transvaal. Similarly, primary centres in Durban, Cape Town and Stellenbosch could serve comparable areas. From such a primary unit services should radiate out to smaller provincial hospitals and after-care clinics.

I believe that surgeons and other skilled staff from the central unit should visit outlying hospitals, where decentralized orthopaedic work could be done with the assistance of local surgeons and a nucleus of full-time staff. The visiting surgeon of such a secondary centre should review and discuss all work done by the local men between visits—this would assist in their training and would disseminate useful techniques to the centres. The full-time men at such a centre should have the status of registrars and should be integrated into a rotating system where a man spends, for instance, 4 years training as a surgeon.

One year of this training could be devoted to adult European orthopaedics, 1 year to European children's orthopaedics, and 1 year to non-European orthopaedics in a major centre where the registrar would get maximal opportunities for applying techniques learnt and doing major work of all types under supervision. In his 4th year he would be competent to undertake all routine work at a secondary centre and would review his work at weekly or fortnightly intervals with the visiting senior surgeon. He would then, after 4 years, be ready to practice on his own, or join a teaching unit, according to his special aptitude.

Remote Areas

In the areas ranging up to 100 miles from the secondary centres it would be wasteful to send surgeons away from institutions where there are adequate nursing, theatre, and X-ray facilities. It would be better and cheaper to bring in the cases to the secondary centres by ambulance or bus. Johannesburg, for instance, would have a primary centre, Klerksdorp a secondary centre, and areas in the far South-Western Transvaal could be served by a decentralized clinic held only once in 3 months by a visiting surgeon from the secondary centre.

At this decentralized clinic the after-care sister, who would know all cases under treatment, would collect about 10-20 cases once a week and arrange for them to go by bus to the secondary centre where all out-patient services could be rendered the same day. In this manner appliances could be maintained, plaster casts changed, X-rays taken, diagnoses made and checked, and continuity of control achieved. In this field the after-care services by social workers and orthopaedically trained nurses could be organized by local committees assisted by local hospital staffs.

THE PATTERN OF THE PROBLEM

In the United States, out of a population of 170 million, there were an estimated 28 million physically handicapped persons including 6 million under 21 years of age. Six million are sufficiently disabled to find it difficult to find employment. Two million require rehabilitation before they can be employed. Each year ¼ million join the ranks of those needing rehabilitation. In the United Kingdom, about 1 million out of approximately 40 million were on the disabled register in 1952.

Of the 64,000 disabled adults in need of rehabilitation in the USA, 42% have orthopaedic disabilities: 1/5th from poliomyelitis, arthritis or osteomyelitis, 2/5ths from accidents, and 2/5ths from congenital and other diseases. In 1924, 23% of cases in orthopaedic institutions in the USA were treated for surgical tuberculosis. In 1951 this figure was down to 1·3%. In 1924 rickets accounted for 8% of crippled children, but in 1957 it accounted for only 1%. Congenital malformations alone account for 21% of crippled children and cerebral palsy for 20%. Among our European population the position is probably comparable, i.e. that congenital deformities and cerebral palsy each account for 1/5th of our cripples.

There are good grounds for hoping that some of these disabilities can be reduced by preventive measures. The early months of pregnancy are important in the structural development of the embryo and most congenital defects are established during this period. If the mother suffers from vitamin or mineral deficiency congenital deformities may occur. Dwarfism may result from lack of iodine, blindness and eye defects from vitamin-B deficiency. X-ray treatment during pregnancy may result in congenital eye defects, sometimes together with mental deficiency. German measles, a mild virus infection, if contracted during the first 8 weeks of pregnancy may cause one or more congenital defects affecting the eyes, heart, hearing or intelligence of the unborn child. These are some of the examples in which prophylactic work lies on the fringe of a comprehensive programme for the care of cripples. This preventive programme covers a tremendous field.

Rehabilitation

The rehabilitation of the physically handicapped is understood to mean their restoration to the fullest physical, mental, social, vocational and economic usefulness. Of the orthopaedically disabled at least 3/4ths can be rehabilitated by providing adequate medical treatment and any prosthesis needed. Special vocational counselling and selective placement in industry may also be necessary.

The remaining 1/4th, however—the severely disabled—require all the resources of society to bring them to the highest degree of self-sufficiency. About 3% can never be rehabilitated to the extent of independence, and provision should be made for them to be cared for either at home or in suitable institutions. The existing provincial hospitals make provision for the treatment of the vast majority of physically disabled so that they may return to a useful life. But in this country there exists a need for at least 2 centres for the seriously disabled.

The Rehabilitation Association for Injured Workmen, a non-profit-making organization under the wing of the Department of Labour, established an organization 3 years ago—namely the Workmen's Rehabilitation Centre in Johannesburg. This organization deals specifically with workmen as defined under the Workmen's Compensation Act and offers comprehensive facilities for the treatment of severe accidents. It renders medical, surgical, physiotherapeutic and occupational therapeutic services as well as socio-economic assistance. In my opinion this organization may with advantage be broadened to accept difficult cases of physical disablement from sources other than accident as well. It could be the nucleus of a great rehabilitation service for the community, especially if it is integrated with postgraduate medical education to attract the best talents in the profession.

Sheltered Employment

In South Africa sheltered employment already provides hope and a useful life for 1,800 European people who are so handicapped that they are not able to hold their own in the open labour market. Some of these facilities have recently, on the advice of the National

Rehabilitation Board, been made available for rehabilitation purposes.

It is generally felt that the time is opportune to create similar facilities for large numbers of the non-European population with a view to restoring their earning capacity and well-being and ultimately reducing the drag on the economy of the country.

Economic Aspects

Nearly 1 out of 5 of the 64,000 persons rehabilitated in 1952 in the United States through the State Federal vocational rehabilitation programme, had been on public assistance rolls, costing the taxpayers 8½ million dollars a year. But as productive members of society, they earned approximately 22 million dollars in the first year after rehabilitation.

The year's earnings of the 64,000 cases was about 115 million dollars, out of which they paid in income tax to the federal government about 10½ million dollars. The rehabilitation of the severely paralysed hemiplegic, paraplegic and quadriplegic patients was so difficult that only about 60% were employable after rehabilitation.

The real economic and social benefits of rehabilitation result from increased self-care that releases hospital beds and frees families and institutions from the care of helpless persons. Morale, self-respect and personal freedom are positive benefits derived by the patients themselves.

Paraplegia

In South Africa, a paraplegia centre is urgently needed, preferably in Johannesburg, because this city has the density of population, the concentration of accidents and the industrial resources to provide opportunities for placement, as well as the skilled medical, nursing and other personnel required. The National Rehabilitation Council may well be persuaded to establish a centre for the apparently hopeless cases of paraplegia which cannot be effectively handled in provincial hospitals.

A coordinated effort of the Provincial Hospital Departments, the Rehabilitation Association for Injured Workmen, and the National Rehabilitation Council, could lead to the development of a unit offering rehabilitation for the most difficult problem-cases in the country.

Voluntary Institutions

These institutions have done pioneer work in the rehabilitation of crippled children. A fair criticism of their activities, however, has been that some of these institutions have supplied hospitalization out of funds collected, whereas hospitalization is really a direct responsibility of the Provincial Hospital Departments concerned.

Furthermore, there is the ever-present danger that parents and families, once relieved of their burden of caring for their own children, will be happy to leave this responsibility to someone else. It is true that severe and prolonged cases of disablement require long-term institutional care; but some voluntary institutions are boarding and lodging cases that could be better cared for in their own homes provided that they could be checked at an orthopaedic clinic at intervals and followed up through the after-care service.

It is my conviction that the main function of cripple-care organizations is to establish the field work—finding the cases, organizing diagnostic clinics, persuading parents to have their disabled children attended to, and ensuring continuity of follow-up until ultimate readjustment is achieved. Money should be more widely spent on organization and on field service than on a possible excess of bricks and mortar.

Education of Crippled Children

To provide educational opportunities for severely disabled children, special provision has to be made, especially in regard to transportation, special equipment, and long-stay convalescent homes and training centres.

Screening of cases for admission and screening of cases during their stay will ensure that services are given to those most in need and that children capable of facing life on equal terms with their fellows are not needlessly estranged from their families by prolonged institutional care.

Training of Personnel

One of the most urgent aspects of cripple-care work is finding suitably trained social workers, sisters for after-care work, and orthopaedic nurses, both European and non-European. Since social work covers such a wide field, we should ensure that a certain proportion of the workers acquire an orthopaedic bias in their training. It is desirable that university degree courses should be broadened in such a way that a period of training in cripple-care work could be incorporated in the curriculum. The country cannot afford to lose orthopaedically trained nurses to fill the gaps in after-care services; but a social worker with a B.A. degree could learn enough of cripple-care work in 3 months to make her a valuable assistant in the field work.

The few orthopaedic nurses trained under the auspices of the National Council for the Care of Cripples are hopelessly inadequate for the needs of the country. Fortunately, the Cape Province has adopted the scheme officially, and there is evidence that the Transvaal Provincial Hospital Department may develop training schools for both European and non-European orthopaedic nurses in the near future.

There is a considerable body of opinion that the training of physiotherapists is badly planned at present. Too much accent is placed on the first 2 years of training which follows the same pattern as a doctor's training. I do not believe that detailed knowledge of anatomy and physiology are necessary in the training of therapists. Uniformity in the training of physiotherapists in the provinces is desirable especially from the point of view of registration by the Medical Council.

Orthopaedic Surgeons

The training of surgeons varies greatly in South Africa. In some hospitals practically self-trained men acquire the right to register as specialists without sufficiently working under supervision during that period. In other institutions, postgraduate training in orthopaedics is inadequate. The universal defect of inadequate training in rehabilitation work for surgeons can only be remedied if more Chairs of Orthopaedics, Traumatology and Rehabilitation are established.

Once again it is stressed that it is the duty and function of the National Rehabilitation Council to further the academic and research aspects of orthopaedic and rehabilitation services so as to provide the country with the skilled personnel we need today and shall require on a still greater scale in the future.

AGSTE TUSSENTYDSE KONGRES VAN DIE SUID-AFRIKAANSE VERENIGING VAN VERLOSKUNDIGES EN GINEKOLOË (M.V.S.A.): BLOEMFONTEIN: 2-5 MAART 1959

EIGHTH INTERIM CONGRESS OF THE SOUTH AFRICAN SOCIETY OF OBSTETRICIANS AND GYNAECOLOGISTS (M.A.S.A.): BLOEMFONTEIN 2-5 MARCH 1959

President Dr. J. C. Coetzee *President* *Vise-President* Prof. L. J. te Groen *Vice President*

Op Sondag 1 Maart 1959 om 8 nm. sal die Jaarlikse Algemene Vergadering van die Suid-Afrikaanse lede van die R.C.O.G., gevolg deur 'n vergadering van die Suid-Afrikaanse Streeksraad van die R.C.O.G., gehou word te Maitland Hotel, Bloemfontein.

On Sunday 1 March 1959 at 8 p.m. the Annual General Meeting of the South African members of the R.C.O.G., followed by a meeting of the South African Regional Council of the R.C.O.G., will be held at the Maitland Hotel, Bloemfontein.

PROGRAM : PROGRAMME

Die Kongresbyeenkomste sal gehou word in die Senaatskamer van die Universiteitsaal van die Universiteit van die Oranje-Vrystaat.

The Congress meetings will be held in the Senate Room in the University Hall of the University of the Orange Free State.

Maandag 2 *Maart* 1959 *March* 2 *Monday*

vm. 8.30- 9.00 a.m.: Inskrywing van lede in die Universiteitsaal
Registration of members at University Hall

Oggendsitting : Morning Session
Voorsitter—Dr. J. C. Coetzee—Chairman

vm. 9.00-10.15 a.m.: Dr. I. E. van den Ende—The anatomy and pathology of premature separation of the placenta.
vm. 10.15 a.m. : Kongresfoto : Congress photograph.
vm. 10.30-11.00 a.m.: Teepouse—Tea interval.
vm. 11.00-12.15 p.m.: Dr. P. M. Bremer—The danger of uterine rupture after Caesarean section.
nm. 12.15 p.m. : Amptelike opening van die Kongres deur Sy Edele mnr. J. J. Fouche, Administrateur van die Oranje-Vrystaat.
Official opening of Congress by the Hon. Mr. J. J. Fouche, Administrator of the Orange Free State.

Middagsitting : Afternoon Session
Voorsitter—Prof. J. Black—Chairman.

nm. 2.30- 3.30 p.m.: Dr. W. P. U. Jackson—Prediabetes in obstetrics.
nm. 3.30- 4.00 p.m.: Teepouse—Tea interval.
nm. 4.00- 5.00 p.m.: Dr. N. Walker—Foetal distress—indication for alibi for Caesarean section.
nm. 6 of/or 7 p.m. : Onthaal deur Burgermeester—Mayoral reception.

Dinsdag 3 *Maart* 1959 *March* 3 *Tuesday*

Oggendsitting : Morning Session
Voorsitter—Prof. E. C. Crichton—Chairman.

vm. 9.00-10.00 a.m.: Dr. S. Shippel—The placenta has a life cycle of 9 months. True or false?
vm. 10.00-10.30 a.m.: Teepouse—Tea interval.
vm. 10.30-11.15 a.m.: Mr. B. C. Murless—Adhesions in pelvic surgery.
vm. 11.15-12.30 p.m.: Dr. P. M. S. Fischer en/and Mr. W. Grundhill—Tracheo-oesophageal fistulae in the newborn.

Middagsitting : Afternoon Session
Voorsitter—Dr. D. F. Standing—Chairman.

nm. 2.30- 3.30 p.m.: Dr. W. P. U. Jackson—Intersex presenting as primary amenorrhoea.
nm. 3.30- 4.00 p.m.: Teepouse—Tea interval.
nm. 4.00- 5.00 p.m.: Dr. M. M. de la Harpe—Die oestrogeen produserende testes-sindroom.
nm. 5.15 p.m. : Prof. O. S. Heyns—Demonstration of decompression at Maternity Home, National Hospital.
nm. 8.00 p.m. : Dinee en dans—Dinner and dance, Bloemfontein Hotel. Drag formeel : Dress formal.

Woensdag 4 *Maart* 1959 *March* 4 *Wednesday*

Oggendsitting : Morning Session
Voorsitter—Dr. R. Theron—Chairman.

vm. 9.00-10.00 a.m.: Dr. F. Daubenton—Maternal mortality in Johannesburg.
vm. 10.00-10.30 a.m.: Teepouse—Tea interval.
vm. 10.30-11.30 a.m.: Dr. N. S. Louw—Some aspects in the treatment of hypertension in pregnancy.
vm. 11.30-12.30 p.m.: Dr. A. J. L. van Rooyen—Effect of symphysiotomy on the sacro-iliac joint.

nm. 2.00 p.m. : Gholf te Gholfklub, Bloemfontein.
Golf at Bloemfontein Golf Club.
nm. 3.00 p.m. : Uitstappie na Mazelspoort vir swem, skuitry en tee.
Outing to Mazelspoort for swimming, boating and tea.
nm. 7.00 p.m. : Vleisbraai en informele dans te Gholfklub, Bloemfontein.
Vleisbraai and informal dance at Bloemfontein Golf Club.

Donderdag 5 *Maart* 1959 *March* 5 *Thursday*
Voorsitter—Dr. J. W. Schabort—Chairman.

vm. 9.00-10.15 a.m.: Dr. S. Shippel—Endometrial patterns in the climacteric and its bearing on therapy.
vm. 10-15-10.45 a.m.: Teepouse—Tea interval.
vm. 10.45-11.45 a.m.: Dr. G. P. Charlewood—Experiences with D'Estes operation for sterility due to tubal occlusion.
vm. 11.45-12.45 p.m.: Dr. D. P. de Villiers—The real facts relating to cochleate uterus.

nm. 2.30 p.m. : Jaarlikse Algemene Vergadering van die Suid-Afrikaanse Vereniging van Verloskundiges en Ginekoloë.
Annual General Meeting of the South African Society of Obstetricians and Gynaecologists.

Let asseblief daarop dat tydelike ere-lidmaatskap van die Klub Ramblers en die Gholfklub Bloemfontein, toegestaan is aan lede van die Vereniging wat die Kongres bywoon.

Please note that members of the Society attending Congress have been granted honorary membership of the Ramblers Club and the Bloemfontein Golf Club.

Spesiale Onthale vir Dames : Special Entertainment for Ladies
'n Spesiale program van onthale sal vir dames, wat hul eggenote vergesel, gereël word.

A special programme of entertainments has been arranged for ladies accompanying their husbands to the Congress.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during December 1958 are gratefully acknowledged:

Met hartlike dank word die volgende skenkings gedurende die maand Desember 1958, erken:

Votive Cards in Memory of:

Mrs. S. F. N. Gie *by* Dr. F. G. Geldenhuys; Mrs. Blake *by* Dr. V. Brink; Dr. J. S. du Toit *by* Dr. P. W. J. Keet; Mr. W. K. Gibson *by* Dr. P. W. J. Keet; Geoff Good *by* Dr. S. Cole; Mrs. Foreman *by* Drs. J. Wolfowitz and B. L. Shaff; and Dr. K. Bremer, Dr. T. Fuller, Dr. F. Fehrsen and Dr. J. Weinberg *by* Dr. H. C. Wykend.

Total Amount Received from Votive Cards £10 19s. 0d.

Services Rendered to:

Dr. D. Girdwood *by* Drs. W. A. Miller, M. J. G. Davies and J. H. Bristow.

Dr. J. G. Louw *by* Drs. T. J. Dry, W. G. Davis and K. Brauer.

Miss C. van Heerden *by* Dr. R. Lane Forsyth.

Dr. L. J. te Groen *by* Drs. I. P. Maas, J. K. Bremer, O. J. Verster, J. M. van Niekerk, C. A. R. Schulenburg, J. F. Dippenaar, T. Fichardt, H. Combrink and Professors S. F. Oosthuizen and H. W. Snyman.

Mrs. C. Edelstein *by* Dr. I. Jacobson.

A member of Dr. T. Theron's family *by* Drs. D. J. van der Heever and S. J. Wicks.

Total Amount Received for Services Rendered £118 18s. 0d.

Donations:	£	s.	d.
Dr. A. Swanepoel	2	2	0
Dr. M. L. Hönigsberger	3	3	0
Cape Western Branch Members Box	4	1	0
Southern Transvaal Branch..	2,500	0	0
Total Donations	£2,509	6	0

Grand Total £2,639 3s. 0d.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL ASSOCIATION OF SOUTH AFRICA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held at Medical House, 5 Esselen Street, Johannesburg, on 8, 9 and 10 April 1959, commencing at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
9 February 1959

MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word te Mediese Huis, Esselenstraat 5, Johannesburg, op 8, 9 en 10 April 1959, aanvang 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
9 Februarie 1959

PASSING EVENTS : IN DIE VERBYGAAN

Dr. B. P. Pienaar, M.B., B.Ch., Physician Superintendent, Weskoppies Hospital, Pretoria, and Deputy Commissioner of Mental Hygiene, has been appointed Commissioner of Mental Hygiene. Dr. Pienaar will take up his appointment on 24 March 1959.

* * *

Dr. A. M. Lamont, M.D., Assistant Physician Superintendent, Weskoppies Hospital, Pretoria, has now been appointed Physician Superintendent, and also Deputy Commissioner of Mental Hygiene.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Tuesday 3 March at 12 noon in the large A-floor lecture theatre, Groote Schuur Hospital, Cape Town. Dr. H. Walton will speak on 'Personality change brought about by intesive group psychotherapy'. All interested are invited to attend.

* * *

The South African Society of Anaesthetists, Cape Western Branch. Dr. G. E. Hale Enderby, who is visiting South Africa under the auspices of the South African Society of Anaesthetists, will deliver a lecture on 'Fluothane and hypotension' on Monday 2 March at 8.15 p.m. in the E-floor lecture theatre, Groote Schuur Hospital, Observatory, Cape. All interested are invited to attend.

* * *

Dr. S. Joel Cohen, M.B., B.Ch. (Rand), F.R.C.S. (Edin.) has been elected a Fellow of the Royal College of Obstetricians and Gynaecologists of Great Britain. Dr. Cohen is the consultant obstetrician at the Queen Victoria Maternity Hospital, Johannesburg; first clinical assistant in the Department of Gynaecology, General Hospital, Johannesburg; and a member of the staff of the Department of Obstetrics and Gynaecology of the University of the Witwatersrand.

* * *

Dr. David Friedberg, M.B., B.Ch. (Rand), M.R.C.O.G., F.C.O.G. (S.A.), has commenced practice as a specialist obstetrician and gynaecologist at 113 Lister Buildings, Jeppe Street, Johannesburg. Telephones: Rooms 22-8427, residence 40-5079.

Dr. David Friedberg, M.B., B.Ch. (Rand), M.R.C.O.G., F.C.O.G. (S.A.), praktiseer nou as spesialis in verloskunde en ginekologie te Lister-Gebou 113, Jeppestraat, Johannesburg. Telefone: Spreekkamer 22-8427, woning 40-5079.

* * *

Mr. Manuel Lunz, M.Ch.Orth. (L'pool), F.R.C.S. (Eng.), orthopaedic surgeon, has commenced practice at 205 Osler Chambers, Jeppe Street, Johannesburg. Telephones: Rooms 22-1778, residence 42-2590.

Dr. Manuel Lunz, M.Ch.Orth. (L'pool), F.R.C.S. (Eng.), ortopediese chirurg, het te Osler-Gebou 205, Jeppestraat, Johannesburg, begin praktiseer. Telefone: Spreekkamer 22-1778, woning 42-2590.

* * *

University of Edinburgh, Sir David Wilkie Research Fellowship in Surgery or Medicine. This Fellowship, of the value of £800-£900 per annum, with a possible allowance for expenses of research, and tenable for 2 years, with a possible extension to 3 years, will be open for award in October 1959. It is open to graduates of any University. The holder will be required to carry out approved research work in surgery and/or medicine in the University, and he must attend the honours class in physiology unless he is already a graduate in physiology or in science. He will be expected to maintain contact with clinical work but the time devoted to it will be restricted to 2 half-days a week. During his tenure the Fellow will not be permitted to study or present himself for examination for any higher diploma in medicine or surgery. Applications must be submitted on a prescribed form. Applications from graduates in South Africa should reach the Director, South African Institute for Medical Research, Hospital Street, Johannesburg, by 1 March 1959, from whom also the prescribed form may be obtained. (An advertisement was published on p. xxxiv of the issue of this *Journal* of 7 February 1959.)

* * *

The Thirteenth International Congress on Occupational Health, the first to be held in the Western Hemisphere, will meet in New York City on 25-29 July 1960, according to an announcement by Dr. Leo Wade, of New York, who is chairman of the Organizing Committee. Several thousand physicians, nurses, industrial hygienists and other delegates from more than 40 countries will attend. With experts from all over the world gathered in New York to discuss and share important knowledge in the fields of occupational injury and disease, the 13th Congress will emphasize the fact that the science of health knows no national boundaries. The theme of the New York meeting will be prevention, rather than cure. Programme participants from the many countries will report on their experiences, the findings of both clinical and laboratory research, and on methods for control of occupational health hazards. Plans already are under way to provide meeting facilities, translation services, accommodation for visitors, and other arrangements. The congresses are sponsored by the Permanent Committee and International Association on Occupational Health, of which Dr. Sven Forssman, of Stockholm, is president and Dr. Enrico Vigliana, of Milan, secretary. All previous meetings have been held in Europe, beginning with the first one in Milan in 1906. The 12th Congress was in Helsinki in 1957.

Dr. Wade said the international meetings on occupational

health are one means of implementing an objective outlined by the President of the USA in his latest State of the Union message: 'A programme of science for peace,' President Eisenhower said, 'might provide a means of funnelling into one place the results of research from scientists everywhere, and from there making it available to all parts of the world. There is almost no limit to the human betterment that could result from such cooperation.'

As modern industry continuously expands its uses of materials and technological techniques throughout the world, new occupational hazards come into being to take their places beside such old and well-known ones as arsenic, mercury and lead. Radioactive materials, for example, are being used to an increasing extent in industry. Findings of research, which will be reported at the 13th International Congress on Occupational Health, will supplement present knowledge on the control and effects on health of exposures to radiation in industry. Dr. Wade pointed out that such man-made hazards among industrial workers can be, and in most instances are being controlled, with resulting benefits in health, increased productivity and economic well-being for the world's population.

NEW PREPARATIONS AND APPLIANCES: NUWE PREPARATE EN TOESTELLE

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Efron, G. (1958): S. Afr. Med. J. 32, 1124.
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Blanz, H. and Spörlein, H. (1957): Münch. med. Wschr., 99, 51.
Graudenz, E. and Pfeil, J. (1956): Med. Klin., 51, 44, 1876.

CORRESPONDENCE : BRIEWERUBRIEK

A CENTRAL MEMORIAL FUND

To the Editor: Some 6 or 7 years ago, following on a suggestion which I made, a Fund was sponsored by the Medical Association of South Africa in memory of the late Dr. H. A. Moffat. In October 1955, subsequent to the death of two outstanding medical personalities (I refer to the late Drs. Linsay-Sandes and Lionel Goldschmidt) I wrote to the *Journal* suggesting that the medical profession in South Africa should have the financial resources available which would enable us to perpetuate the names of those colleagues, who, by virtue of outstanding and distinguished service rendered to the profession, either in the academic sphere or in the public activities of the Medical Association, deserve to be thus commemorated.

Once again, may I be permitted to urge on those responsible the need for the establishment of such a Memorial Fund. If a small levy (5 or 10 shillings a year) were imposed on all members of the Association and the proceeds devoted to a 'Memorial Fund', within a matter of a few years a substantial amount would become available to provide scholarships, prizes, etc. at the various medical schools in the Union.

During the past few years several outstanding leaders in the profession have passed on and nothing whatsoever has been done to commemorate their names. A central fund, controlled by the Medical Association of South Africa, would avoid this sad neglect of those who served the profession so faithfully and so long. 'Lest we forget'.

Enclosed please find my donation to the Dr. J. S. du Toit Memorial Fund.

Jack Abelsohn

611 Medical Centre
Cape Town
9 February 1959

STEROID THERAPY AND OPERATIVE COLLAPSE

To the Editor: Anaesthetists and surgeons are becoming increasingly aware of the problem of collapse during operation due to adrenal cortical suppression in patients who are having, or have had, corticosteroid therapy. While there is wide individual variation in the duration of the adrenal suppression after cessation of treatment, there have been cases of irreversible shock following even minor surgical procedure in patients whose steroid therapy had ceased 24 months previously.

Special pre-operative preparations for such patients can prevent these disasters, *provided* the patient is aware that he has had steroid therapy.

The purpose of this letter is to urge all practitioners, when prescribing a course of ACTH or any corticosteroid, to warn patients, as a routine, of the necessity of advising the anaesthetist and surgeon if, and when, surgery is necessary. This should become as much an automatic procedure by practitioners as the relevant advice given in cases of diabetes, allergy, drug sensitivities, etc.

Dennis Glauber

17 Edgewood Avenue
Birdhaven, Johannesburg
2 February 1959

IMMUNIZATION OF CHILDREN

To the Editor: In your column 'Questions Answered'[1] in the *Journal* of 10 January some excellent advice is given. However, there are two items in which the recommendation may give rise to misunderstanding. In the paragraph on contra-indications to poliomyelitis vaccine it is stated that pregnancy is a possible contra-indication. I am not sure what contingency the writer had in mind, but as pregnancy is one of the factors concerned in provoking paralytic attacks in individuals who otherwise may have had a silent infection, pregnant women were given first priority for poliomyelitis vaccine in the United States when it was in short supply. They were also given some priority in South Africa. Current authoritative opinion favours the giving of vaccine to non-immune pregnant women to lessen their liability to paralytic poliomyelitis, should they become infected.

In the same paragraph, in dealing with allergy, there is a sentence which states 'even frank poliomyelitis may occasionally occur'. This too, may be misunderstood for, of course, frank poliomyelitis should never result from poliomyelitis vaccine given to allergic individuals. It is possible that the inoculation may serve as a provocative irritation in individuals already infected with poliovirus, and therefore it is recommended that poliomyelitis vaccine is given at the time when poliomyelitis is least prevalent in those countries where there is a clear seasonal incidence. However, this risk appears to be very slight and is now largely ignored. Appropriate precautions should be taken when giving vaccine to allergic individuals, especially if they are known to be hypersensitive to penicillin, as described by your contributor.

J. H. S. Gear
Director of Research

The Poliomyelitis Research Foundation
P.O. Box 1038
Johannesburg
9 February 1959.

1. Questions Answered (1959): S. Afr. Med. J., 33, 37.